

Severity of dental fluorosis on the Dean scale

The level of fluoride and IQ in different group by dental fluorosis

Group	No.	Water F	IQ	Urine F	Serum F
0	301	0.50±0.53	99.76±3.50	1.13±0.71	0.044±0.017
1	65	1.88±1.07	94.18±13.77	2.70±1.15	0.071±0.023
2	59	2.44±0.66	93.27±13.10	3.69±1.61	0.082±0.016
3	63	2.67±0.63	91.51±12.84	3.85±1.79	0.085±0.019
4	24	2.89±0.81	95.33±14.64	3.81±1.80	0.084±0.018

Xiang's presentation at FAN conference , Sept 6, 2014

From: [Gerald Steel](#)
To: [Phillips, Theresa \(DOH\)](#)
Cc: [Audrey Adams](#); [Scott Shock](#); [Bill Osmunson](#)
Subject: WAC 246-290-460 Rulemaking - Recent information on Harms of Fluoridation
Date: Tuesday, February 23, 2016 11:11:50 AM
Attachments: [Harms of Fluoridation by Gerald Steel 3-26-15.pdf](#)

I submit this comment on behalf of myself and King County Citizens Against Fluoridation.

The attached document, prepared eleven months ago, presents some of the recent publications on the harms of fluoridation. It shows that there is a significant correlation of increased diagnosed ADHD prevalence with increased levels of fluoridation in the 50 states based on government statistics. Studies have recently become available that explicitly find reduced average IQ in children who drink water at 0.7 to 1.2 ppm fluoride compared with neighboring children who drink low fluoride water. We cannot ignore the 44 human studies that show reduced IQ in children correlated with increased fluoride ingestion mostly from drinking water. Protect the children. Put a moratorium on water fluoridation. Fulfill the SBOH obligation in RCW 43.20.050(2) and assure that public drinking water is safe. Do not let a statistically insignificant claim of reduced tooth decay trick you into supporting unsafe fluoridated drinking water. Protect the children.

Thank you for consideration of our comments.

Gerald Steel
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Tel/Fax (360) 867-1166



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

OFFICE OF
GENERAL COUNSEL

February 14, 2013

Gerald Steel, PE
7303 Young Road NW
Olympia, WA 98502

Dear Mr. Steel:

This is in response to your letter of December 28, 2012 to EPA Administrator Lisa Jackson in which you asked several questions about the status of an MOU between EPA and the Federal Drug Administration (FDA) published in 1979. I am replying on behalf of her.

Your first question is whether, from the viewpoint of EPA, the purpose of a 1979 Memorandum of Understanding (MOU) between EPA and the Federal Drug Administration (FDA) was "to take away from FDA, and give to EPA, responsibility for regulating public drinking water additives intended for preventative health care purposes and unrelated to contamination of public drinking water?" Your second question is whether, if that was the purpose of the 1979 MOU, the MOU was terminated through a subsequent Federal Register notice.

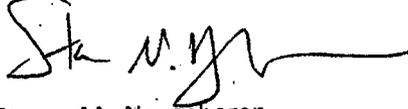
The answer to your first question is no, so there is no need to address your second question. The purpose of the MOU was not to shift any responsibilities between the Agencies. Rather, it was to help facilitate effective coordination of our respective legal authorities. Under the Safe Drinking Water Act (SDWA), EPA is the lead federal agency with responsibility to regulate the safety of public water supplies. EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride, other than to limit the addition of such substances to protect public health or to prevent such substances from interfering with the effectiveness of any required treatment techniques. SDWA Section 1412(b)(11); see also A Legislative History of the Safe Drinking Water Act, Committee Print, 97th Cong, 2d Session (February 1982) at 547. The Department of Health and Human Services (HHS), acting through the FDA, remains responsible for regulating the addition of drugs to water supplies for health care purposes.

The 1979 MOU was intended to address contamination of drinking water supplies as a result of direct or indirect additives to drinking water, not to address the addition of substances solely for preventative health purposes. 44 Fed. Reg. 42775 (July 20, 1979) ("EPA and FDA agree: (1) that *contamination* of drinking water from the use and application of direct and indirect additives and other substances poses a potential public health problem...")(emphasis added). It was intended to avoid potentially duplicative regulation of "food", which FDA had, in the past, considered to include drinking water. 44 Fed. Reg. 42775 (July 20, 1979). The MOU did not address drugs or other substances added to water for health care purposes.

Gerald Steel, PE
February 14, 2013
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I hope that this has adequately answered your inquiry. Please do not hesitate to contact Carrie Wehling of my staff (202-564-5492) if you have further questions about this.

Sincerely,

A handwritten signature in black ink, appearing to read "St. M. Neugeboren", with a long horizontal flourish extending to the right.

Steven M. Neugeboren
Associate General Counsel
Water Law Office

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10

1200 Sixth Avenue, Suite 900
Seattle, WA 98101-3140

OCT 10 2012

OFFICE OF
WATER AND WATERSHEDS

Mr. Gerald Steel, PE
Attorney at Law
7303 Young Road NW
Olympia, Washington 98502

Dear Mr. Steel:

Your letter dated August 3, 2012, has been forwarded to the Office of Water and Watersheds for a response because my office is responsible for the implementation of the drinking water regulations. In your letter, you reiterate certain provisions of the Safe Drinking Water Act as we described them in letters from our office dated April 7, 2011, and November 17, 2011.

You go on to refer to various sections of the Washington Administrative Code, specifically WAC 246-290-220(3), which addresses treatment chemicals added to drinking water and WAC 246-290-460, which addresses drinking water fluoridation practices.

As noted in the U.S. Environmental Protection Agency (EPA) letter of November 17, 2011, neither WAC 246-290-220(3) nor WAC 246-290-460 are related to the requirements of the Federal Safe Drinking Water Act in Washington State.]

You ask if there is any law, regulation, or directive giving the EPA authority to prevent the Food and Drug Administration and/or Health and Human Services from exercising their drug authority to make a finding that fluoride products added to drinking water are drugs and if there is any law, regulation or directive giving the EPA authority to reverse any FDA regulatory action resulting from such a finding. The answer to both of these questions is no. The EPA has no authority to intervene in the actions of these agencies. If you have additional questions, please contact Fredianne Gray, our Regulatory Fluoride expert, at (206) 553-6387.

Sincerely

Daniel D. Opalski
Office of Water and Watersheds

My Views on the Fluoridation of Water

Robert L. Isaacson
Distinguished Professor of Psychology
Binghamton University

A note on terminology: **Fluorine** is an element in the halogen group as are chlorine and iodine. Of all the known elements, fluorine is the most chemically reactive, most powerful oxidizing, and most electronegative element. It is a stronger oxidizing element than ozone. It reacts with many compounds at room temperature. It is never found in its pure form in nature.

Fluoride: Any combination of fluorine with another element or chemical group of elements. Thus, the addition of fluorides to the drinking water can indicate the addition of a large number of chemical agents. The most commonly used fluorides for this purpose are sodium fluoride, NaF, and compounds that contain both fluorine and silicon. Such agents are collectively called "Fluorosilicates." They include fluorosilicic acid, fluorosilicate, hydrofluosilicic acid, and hexafluorosilicic acid.

In 2003 when I accepted an invitation to join the National Research Council's Committee formed to evaluate the EPA standards for the amount of fluoride that should be allowed in our drinking water, I had no fixed opinion on whether or not fluoride should be added to drinking water. Probably I was asked to serve on the committee because I had organized a series of experiments published between 1993 and 1998 using rats to study the effects of chronic administration of aluminum fluoride in their drinking water. My primary interest was in the effects of aluminum on the brain and behavior. Aluminum fluoride was used because fluoride facilitates the passage of aluminum into the brain. At the time, aluminum was considered by a number of scientists to be an important factor in Alzheimer's disease. Scientists are still actively investigating this possibility. Our studies had to include the investigation of the effects of the fluoride since the aluminum and the fluoride readily become associated after ingestion. In essence we wanted to know the effects of the aluminum, fluoride, and the aluminum-fluoride complex.¹

In my more than three years working on the National Research Council Committee I learned about the many influences fluoride has on the nervous system and the brain. I also learned about the variety of ways in which people become exposed to it and the work that had been done in trying to determine if fluorides were a hazard to human health and well being. The results and recommendations of this Committee were published late in 2006.² Slowly, I came to the conclusion that there were strong experimental and clinical indications that fluorides present health hazards to people in many ways. The more I learned, the more I became convinced that the addition of fluorides to drinking water was, and is, a mistake. Accordingly, I decided to share some of my conclusions with any who might wish to know them.

Fluorine-containing compounds can affect every living animal and person. Exposure to fluorides can come from the air, the water, and the foods we eat. Fluoride compounds were long used as insecticides. They were especially effective for ants and roaches. Their containers were always boldly marked as a poison and there were warnings on the label to keep them well away from children. This is mentioned only to note that for many years fluorides have been considered to be major health hazards.

In regard to health the total accumulation of fluorine in the body is important. Only about half of the amount of fluorides taken in by a person is excreted. The rest stays in the body. Toxic effects are determined by the amount of fluoride stored in the body, current exposure level, and age at the time of exposure. In addition each person has his or her own tolerance level for fluorides. Once this level is exceeded however, dysfunctions of body and/or brain will occur. How these dysfunctions will be expressed depends on the genetic makeup and past experiences of the person. Another factor

that helps determine a person's sensitivity to fluoride is their age. Both the very young and the very old are most likely to be adversely affected after exposure to fluorides.

As noted, different people exhibit a wide range of toxic reactions to fluorides. Some people affected by fluorides complain of general weakness and chronic fatigue, others complain of cramp-like pains in the abdomen, or nausea. Still others express toxin-induced effects by diminished vision, headaches, migraine attacks, or pains in muscles and joints. These fluoride effects have been described in books by Leo Spira (1950, 1959)³ and George Waldbott and his associates (1978).⁴ It is difficult to determine whether or not a given set of symptoms is a consequence of fluoride intake. It is first necessary to rule out the presence of other diseases that could produce the observed symptoms. A correct diagnosis is best shown by repeated observations of an individual when drinking pure water or water contaminated with a fluoride. These exposures must last for periods of a week or two under conditions in which the patient doesn't know which type of water is being consumed. If the symptoms disappear when the person is drinking pure water and return with the resumption of drinking the fluoride-treated water, this is evidence that the problems arise from the fluoride. Leo Spira and George Waldbott and his associates used this type of experimental approach in their research.

Since people vary so much in their sensitivities to fluorides and also in the nature of their symptoms caused by this toxin, determination of a uniform "safe" level of exposure for everyone is impossible. In a way, fluorides are like ozone: there is no really "safe" level that would protect everyone. The Congressional Safe Drinking Water Act instructed that the level of fluoride in drinking water should be set so as to be safe for *everyone*

regardless of age or overall health.

Increasing the problems that can be induced by fluorine in its different forms is its ability to enhance the effects of other toxins to which we are exposed. For example, fluorides in the drinking water accelerate the absorption of lead, aluminum, and silicon into the body and brain.

The toxic effects of lead have been known for hundreds of years. In recent years the focus of attention has been on the learning deficits lead produces in children. The mechanisms proposed for the induction of this effect are not known entirely but there is evidence that many of the most important neurotransmitters of the brain are being affected. These include alterations in dopaminergic, cholinergic, and glutaminergic systems as well as in the "supportive" glia cells of the brain. There is also evidence that lead toxicity may go beyond impairments of intelligence. Indeed, lead toxicity may produce behavioral changes that include loss of impulse control and a related increase in the frequency of violent acts.⁵

The health hazards associated with enhanced incorporation of lead are not induced by all fluorides but primarily, and maybe only, by the addition of a silicofluoride to our drinking water. The fluoride most often added to our drinking water is hexafluorosilicic acid. This fluorosilicate dissociates when it enters the body. One component contains silicon and another fluorides. As a consequence when silicofluorides are added to our drinking water there are really two toxic hazards: one coming from the fluoride and another from the silicon. Silicon can produce its own toxic effects including the formation of solids (silica and silicates) that can lodge anywhere in the body. In addition the silicon portion also can generate destructive hydroxyl ions in many organs including the brain. The brain damage caused by the production of these free radicals has been related to anti-social behavioral

actions and violence.⁶ Recently data from 327 towns and cities, some having fluoridated water and others not, have been compared in terms of crime rates. All the communities with fluoridated water had substantially higher rates than did those with non-fluoridated water. This indicates that fluorides can act to enhance the damage being done by other toxins.

The impairment of intelligence from lead toxicity is now well established. It is possible that fluorides can produce negative effects on measured intelligence also. The country devoting the greatest attention to this possibility is China.

As of February 2007, several groups of Chinese investigators had published over 20 scientific papers on this topic. Scientists from many different areas of China participated in these investigations. The children studied in these reports ranged in age from 4 to 14. All were tested by the same or very similar standardized I.Q. tests. Overall the results came from children tested at different places, at different ages, and tested by different investigators. All the results from China have found that communities with high levels of fluoride in their drinking water have fewer children scoring at the "bright" end of the intelligence spectrum than communities with low or no level of fluoride. Since China does not fluoridate their drinking water, the Chinese studies compare the I.Q. scores of children from towns and school areas that differ in the amount of fluoride naturally present in their water supplies. While not all of Chinese studies were perfectly designed, the large number of studies showing the same pattern of results calls for our attention. A negative effect of fluoride on intelligence seems to be a possibility.

Other studies in China have indicated that fluoride exposure in the drinking water of mothers during the 6th to 8th months of pregnancy can

produce anatomical changes in the fetal brains. There are also reports of impaired responsiveness to visual and auditory stimuli in babies in the first three days after birth induced by the intake of fluoridated water by young mothers during gestation.⁷

The ingestion of fluoride tends to increase the uptake of aluminum by the brain. In the studies done in my laboratory the increase in aluminum in the brains of rats was *not* a function of the amount of aluminum fluoride given the animals in their drinking water. The smallest dose of aluminum fluoride produced about the same amount of aluminum in the brain as a dose 10 or even a 100 times larger. A small amount of fluoride seems capable of opening aluminum pathways to a maximal degree. It is of great interest that the relative risk of having Alzheimer's disease is increased when individuals had high amounts of aluminum in the brain coupled with low amounts of fluoride.⁸ Another observation of interest is that aluminum by itself may not exert toxic effects on the nervous system. It may only become a toxin after joined together with a fluoride to become an aluminum fluoride.⁹

The chronic administration of fluorides in rats produces changes in the microscopic structure of the brain. There were significant losses of cells in areas of the hippocampus and the neocortex. Many apparently dead or dying cells were found in areas analogous to locations in which similar dying cells are found in the brains of Alzheimer's patients.

A common and, perhaps universal, characteristic of dementia is a reduction of aerobic metabolism in the brain. The blood supply reaching the brain is the primary supplier of oxygen and nutrients. Reductions in this sole source of brain energy can be due to a number of physical or chemical changes. When the brains of animals chronically exposed to aluminum fluoride were examined histologically, deposits of aluminum-based crystals

were found along the walls of both large and small blood vessels in the brain. Similar deposits were also found in the center of many vessels suspended by collagen fibers. These deposits decreased the normal transfer of oxygen from the red blood cells to the brain since they must have created turbulence in its blood flow. It is of historical interest that Alois Alzheimer, the man for whom a type of dementia was named, noted that most patients with this disorder suffered from atherosclerosis in addition to other brain anomalies. This condition is one in which there are deposits formed on the sides and in the center of arteries in the brain. The deposits disrupt the flow of blood to the brain often cause severe brain damage.

Brain functions are entirely dependent on the availability of oxygen. The brain itself consumes 20% of all the oxygen used by the entire body. The brain area most affected by the reduction in oxygen availability is the forebrain. The lower centers of the brain, namely the midbrain and hindbrain, are more resistant to oxygen deprivation. This is why the higher functions of the brain are the first to be affected, as well as the most affected, by oxygen deprivation. Basic motor and visceral functions are often spared even in patients with profound interruptions of normal blood supplies to the brain.

One of the best-known chemical alterations produced by fluorides is a reduction in cholinesterases, including acetylcholinesterase. Fluorides also directly affect the actions of many of other important neurotransmitters in the brain. Fluorides seem to have a special attraction to acetylcholine. Nerve cells that synthesize this transmitter have numerous projections to many forebrain areas, including the neocortex and deeper areas of the brain that provide information to the neocortex.

Not only do fluorides change the amount of the acetylcholine in the

brain, they selectively block certain receptors that respond to this transmitter. Fluoride reduces the number of one type of "nicotinic receptors" for acetylcholine. Some other nicotinic subtypes are not affected.¹⁰ Added to all of the other alterations in structure and function of the brain caused by fluorides, the opportunity for mental and behavioral changes are almost limitless.

While the cholinergic system of the brain has been most studied in regard to the effects of fluoride, it is not the only neural transmitter affected. It is likely that all neural transmitter systems are affected by fluoride intake, directly or indirectly. Other anomalies related to fluoride intake are found in many other chemical systems of the brain.

During the period from 1956 to 1963, the endocrinologist, Ionel Rapaport, presented evidence of a link between fluoride exposure and the numbers of babies born with Down's syndrome, (Trisomy 21). For a number of years the only follow up to his work was in the form of epidemiological comparisons between the number of births of such children both to mothers living in fluoridated drinking water vs. the number of such born to mothers births in or non-fluoridated drinking water areas. The demographics of the two or more areas being compared were not fully taken into account in most of the studies. Maternal ages were also not taken into consideration. Overall, the "follow up" studies to Rapaport's report were not decisive but none of them failed to rule out his original findings.

Furthermore, a determination of fluoride effects using standard epidemiological procedures cannot provide convincing information. This is because it is impossible to find populations virtually the same in all regards except for the amount of fluoride in their drinking water. Another problem arises from the difficulty in accurately determining the number of Down's

syndrome children born. Some investigators use the number of birth certificates on which the attending physician notes that the baby had Down's syndrome. Other investigators use only closed hospital records made sometime later. Still other investigators use both. Neither method is perfect. The use of entries on hospital records would seem to be the most accurate method since physicians seldom enter the nature of possible deformities like Down's syndrome on birth certificates after delivery. Indeed because of the possibility of making a mistake from delivery, the diagnosis is not often made until a determination can be made by laboratory results.

Probably the best collection of relevant data comes from a study of births of children born in two areas of Atlanta, Georgia, as reported by Erickson et al. in 1976. Two different estimates of the number of Down's children and normal children were presented. One estimate of Down's syndrome births was made by the examination of copies of birth certificates and the other was based on hospital records. A re-examination of Erickson's data by Burgstahler¹¹ showed an overall enhancement of Down's syndrome births to mothers from the fluoridated area. Later, in 1998 Takahashi did a fine grain analysis of data from a number of sources that included the corrected numbers from the 1966 Erickson report.¹²

In the Takahashi report a clear-cut relationship between fluoride exposure and the number of affected children was found in mothers 30 years of age and younger. Recently, Juan C. Molino¹³ and I using only data from hospital records found the same age-fluoride-Down's syndrome birth effect.

In his report Takahashi extended the analysis of his data through the use of a regression analysis. He wanted to determine if there could be any dose that would not increase the likelihood of having a Down's syndrome child. According to his calculations there was no such dose. All doses of

fluoride caused some enhancement of the likelihood of a woman having such a child. There are other data supporting the idea that fluorides can induce genetic alterations. Evidence indicating biochemical interactions of fluoride with the genetic mechanisms of cell division are presented in the NRC report on Fluoride in the Drinking Water. (See Endnote 2)

When the possible benefits and possible dangers of fluoride are considered there really is no comparison. Consider the following: There is no known benefit of adding any form of fluoride to our drinking water. Who would want to increase chances of having a less than perfect child? Who would wish to take a chance on a possible reduction of their own mental capacity? Who would want to have their personality altered by fluoride induced alterations in their brain chemistry? Who would want to increase their odds of developing Alzheimer's disease? Eliminating the addition of fluoride to our drinking water would remove these possibilities. The cost of doing this is zero. In fact it would enrich the communities now adding fluorides to their drinking water.

Endnotes

1. Varner, J. A., Huie, C. W., Horvath, W. J., Jensen, K. F., and Isaacson, R. L. (1993) Chronic AlF_3 administration: II. Selected histological observations. *Neurosci. Res. Comm.* 13:99-104. Varner, J. A., Jensen, K. F., Horvath, W. J. and Isaacson R. L. (1998) Chronic administration of aluminum fluoride or sodium fluoride to rats in drinking water: alterations in neuronal and cerebrovascular integrity. *Brain Res.*, 784: 284-298. Varner, J. A., Horvath, W. J., Huie, C. W., Naslund, H. R., and Isaacson, R. L. (1994) Chronic aluminum fluoride administration. *Behav. Neural Biol.*, 61: 233-241. Isaacson, R. L., Varner, J. A., and Jensen, K. F. Toxin-induced blood vessel inclusions caused by the chronic administrations of aluminum and sodium fluoride. *Ann. NY Acad. Sci.*, 825:152-166.
2. The final report of the committee was published by the National Academies Press in December 2006, entitled "Fluoride in drinking water." It can be obtained from the National Academies Press and by special order from any bookstore. The electronic link to the NRC/NAS publication sites:

<http://nap.edu/catalog/11571.html>.

3. Spira, L. The drama of fluorine, archenemy, of mankind. Milwaukee: The Lee Foundation for Nutritional Research, 1950, 1959.
4. Waldbott, G. L. Fluoridation the great dilemma, Lawrence, KA: Coronado Press, 1978.
5. Masters R. D., Coplan, M.J. Association of silicofluoride treated water lead with elevated blood lead. *Neurotoxicology*, 2000. 21:1091-1100. Masters, R. D., Coplan M. J. A dynamic, multifactoral model of alcohol, drug abuse and crime: Linking neuroscience and behavior to toxicology. *Soc. Sci. Information*, 1999, 38: 591-624.
6. Seavy, J., (2005) Water fluoridation and crime in America. *Fluoride*, 38:11-22.
7. Du Li. (1992) The effect of fluorine on developing human brain. *Chinese Journal of Pathology*, 21:218-20. Li Jing, Yao L., Shao, Q-L, and Wu, C-Y. (2004) Effects of high fluoride level on neonatal neurobehavioral development. *Chinese Journal of Endocrinology*, 23: No.5.
8. Belovjovic, G., Jakovlevic, B. (1999) Aluminum and Alzheimer's disease. *Spr. ArArh. Celok* 126: 283-289.
9. Strunecka, A. (1999) Aluminum plus Fluoride: a new deadly duo. *Dement.* 1: 2-3.
10. Long, Y-G, Wang, Y-N, Chen, J., Jiang, S-F, Nordberg, A., and Guan, Z-Z. (2002) Chronic fluoride toxicity decreases the number of acetylcholine receptors in the rat brain. *Neurotox. Terat*, 23: 751-757.
11. Burgstahler, A. W. (1966) Fluoridated water and Down's syndrome. Long abstract of a report of the 21st Conference of the International Society for Brain Research, Budapest.
12. Takahashi, K. (1998) Fluoride-linked Down syndrome births and their estimated occurrence due to water fluoridation. *Fluoride*, 31: 61-73.
13. Juan Carlos Molina is the Director of the Ferryra Research Institute at the University of Cordoba, Argentina, as well as holding his distinguished professor position there. He also is a visiting research professor at Binghamton University.

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not intended to provide medical advice but rather for the sharing of knowledge and opinions of the author. Decisions about health advice should be based on a personal one-on-one basis with an appropriate physician.

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STATE OF WASHINGTON
DEPARTMENT OF HEALTH
DIVISION OF ENVIRONMENTAL HEALTH
PO Box 47820 • Olympia, Washington 98504-7820

October 28, 2008

Dr. Eloise Kailin, MD
P.O. Box 1677
Sequim, WA 98382

Dear Dr. Kailin:

At the October 21, 2008 meeting of the Clallam County Board of Health you raised the question of whether or not the product used by the city of Port Angeles to fluoridate the city's water supply meets the regulatory requirements of the Washington State Department of Health. In follow-up we have confirmed that the city uses fluorosilicic acid provided from J. R. Simplot Company in Rock Springs, Wyoming. The product is NSF Standard 60 certified and does meet the requirements of our regulations.

At the Department of Health we do not have the resources that would allow us to do independent evaluations of water treatment products. As such we rely on national certification protocols to ensure the safety of water additives. Specifically, Washington Administrative Code 246-290-220 (3), requires that: "Any treatment chemicals, with the exception of commercially retailed hypochlorite compounds such as unscented Clorox, Purex, etc., added to water intended for potable use must comply with ANSI/NSF Standard 60. The maximum application dosage recommendation for the product certified by the ANSI/NSF Standard 60 shall not be exceeded in practice." Since the fluoridation product being used by the city of Port Angeles is certified under NSF Standard 60, the city's use of this product is in compliance with state law.

Attached is a July 2000 letter from Stan Hazan, general manager of the NSF Additives Certification Program, to US Representative Ken Calvert providing information on the NSF program. I hope you find this additional information useful.

Sincerely,

Gregg L. Grunenfelder, Assistant Secretary

Cc: Mary Selecky, Secretary of Health
Tom Locke, Clallam County Health Officer
Denise Clifford, Director Office of Drinking Water

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§ 141.62. Maximum contaminant levels for inorganic contaminants.

Code Of Federal Regulations

Title 40. Protection of Environment

Chapter I. ENVIRONMENTAL PROTECTION AGENCY

Subchapter D. WATER PROGRAMS

Part 141. NATIONAL PRIMARY DRINKING WATER REGULATIONS

Subpart G. NATIONAL PRIMARY DRINKING WATER REGULATIONS: MAXIMUM CONTAMINANT LEVELS AND MAXIMUM RESIDUAL DISINFECTANT LEVELS

Current through November 5, 2015

§ 141.62. Maximum contaminant levels for inorganic contaminants

- (a) [Reserved]
- (b) The maximum contaminant levels for inorganic contaminants specified in paragraphs (b) (2)-(6), (b)(10), and (b) (11)-(16) of this section apply to community water systems and non-transient, non-community water systems. The maximum contaminant level specified in paragraph (b)(1) of this section only applies to community water systems. The maximum contaminant levels specified in (b)(7), (b)(8), and (b)(9) of this section apply to community water systems; non-transient, non-community water systems; and transient non-community water systems.

Contaminant	MCL (mg/l)
(1) Fluoride	4.0
(2) Asbestos	7 Million Fibers/liter (longer than 10 [MICRO]m).
(3) Barium	2
(4) Cadmium	0.005
(5) Chromium	0.1
(6) Mercury	0.002
(7) Nitrate	10 (as Nitrogen)
(8) Nitrite	1 (as Nitrogen)

A-22

SUMMARY OF KEY HARMS FROM FLUORIDATION

By Gerald Steel (geraldsteel@yahoo.com)

3-26-15

EXECUTIVE SUMMARY

The major dietary source of fluoride for most people in the United States is fluoridated drinking water. NRC (2006) at 24 (<http://www.nap.edu/catalog/11571/fluoride-in-drinking-water-a-scientific-review-of-epas-standards>). Currently, local politicians, generally with no medical training, decide whether or not to put fluoridation chemical additives into public drinking waters. HHS and FDA admit that these additives and fluoridated waters are intended for use to prevent tooth decay disease but they refuse to exercise responsibilities under the Food Drug and Cosmetic Act (FDCA) to regulate these articles as drugs. 21 USC 393(a) and (b); 21 USC 321(g)(1). FDA states that the Safe Drinking Water Act (SDWA) relieves it of this responsibility. HHS Dr. Wanda Jones 11-21-14 Letter to Ms. McElheney. EPA administrates the SDWA and so has agency authority for its interpretation. EPA interprets the SDWA to not relieve HHS and FDA of their responsibilities "for regulating the addition of drugs to water supplies for health care purposes." Steven Neugeboren 2-14-13 Letter to Mr. Steel. However, EPA remains responsible for regulating total fluoride in public drinking water through setting a Maximum Contaminant Level (MCL) Goal and setting and enforcing a MCL. This Goal is required by the SDWA to be "set at the level at which no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety." 42 USC 300g-1(b)(4)(A).

In the materials below, I discuss some of the substantial evidence that connects fluoridation to "known or anticipated adverse effects on the health of persons." Generally in the United States fluoridation levels are about 1 mg/L fluoride. There is substantial evidence of harm. With a common margin of safety of 10, safe fluoride levels in drinking water can be no higher than 0.1 mg/L (and must be less because there is fluoride in the diet). There will be no dental caries reduction benefit at 0.1 mg/L fluoride. Therefore, there is no point in adding fluoride to get 0.1 mg/L fluoride. Fluoridation should end. Scientific studies of the mechanisms by which fluoride causes harms should be continued. But there is enough information to know that some subpopulations are harmed by fluoridation, and would be, even if it were reduced to 0.7 mg/L fluoride. So I believe that it is most important to educate the public by developing graphs that show harms and benefits (if any) of fluoridation in the United States. I include graphs of prevalence of Mental Retardation (MR) (Appendix A-1 hereto) and Attention Deficit Hyperactivity Disorder (ADHD) (Appendix A-2 hereto) versus percent of state population fluoridated in the fifty states. These graphs show increasing levels of developmental disabilities with increased percent of population fluoridated. We provide a graph (Appendix A-3 hereto) plotted by Dr. Osmunson DDS) of prevalence of children with good/excellent teeth versus percent of state population fluoridated. This graph shows no increase in children with good/excellent teeth with increased percent of population fluoridated in the fifty states.

What science or ethics-based issues regarding fluoridation are of concern?

● Developmental Disabilities

Impact of population-wide levels of exposure to fluoride on neurodevelopment

I am aware of NIEHS Project # R01ES021446 regarding Prenatal and Childhood Exposure to Fluoride and Neurodevelopment by Howard Hu at the University of Toronto. This project is studying the impact of population-wide levels of exposure to fluoride on neurodevelopment. His pilot research of 40 mother/child pairs found increases in pregnant mother fluoride exposure resulted in lower offspring IQ. (See <http://grantome.com/grant/NIH/R01-ES021446-04>) This is an adverse effect of fluoridation on the mental health of persons. The full study is also looking at impacts of childhood fluoride exposure on neurodevelopment. This study started in June 1, 2012 and ends on Feb. 28, 2017. This study measures fluoride exposure using archived urine, fasting plasma, and toenail specimens. Results from five statistically significant IQ studies (Appendix A-4 hereto from Connett Presentation, Sydney Australia, 2-21-15 (Connett (2015) based on NIEHS publication at <http://ehp.niehs.nih.gov/wp-content/uploads/2012/09/ehp.1104912.pdf> references) already suggests that each increase of fluoride of 0.25 mg/L in drinking water by water fluoridation could lower child IQ by one point. Appendix A-1 hereto, plotted by Dr. Osmunson DDS, shows number of Mental Retardation Children 6-17 years old per 10,000 in the fifty states increases with increasing percentage of state population fluoridated. Appendix A-5 hereto from Connett (2015) shows average IQ reduced about 6 points even when dental fluorosis was Dean Index 1 (very mild) and Dean Index 2 (mild). So it appears that significant IQ loss from fluoridation can occur even with very mild and mild levels of dental fluorosis.

Correlation of fluoridation prevalence on ADHD in fifty states

Appendix A-2 hereto shows a correlation of fluoridation prevalence with Attention-Deficit Hyperactivity Disorder (ADHD) in fifty states. This graph is adapted from Malin (2015) by adding color. (See <http://www.ehjournal.net/content/14/1/17/abstract>) This graph shows percent of children 4-17 medically-diagnosed with ADHD increases linearly with increases in percent of state population fluoridated. Fluoridation information is from CDC. ADHD rates are from the National Survey of Children's Health. Socioeconomic status is controlled. In 2011, 8.8 percent of children in non-fluoridated states were diagnosed with ADHD. This increased to 13.9 percent for fully-fluoridated states. This is a 58% increase. Child ADHD prevalence is linearly correlated with fluoridation prevalence with relatively little scatter.

From the Office of Children's Health Protection (OCHP) of EPA, Children's Environmental Health Facts show concerns for "Developmental Disabilities." This webpage states that between 3 and 8 percent of children will have developmental disorders such as ADHD or mental retardation. The data presented above shows medically-diagnosed ADHD levels actually averaged 11 percent in 2011. This data alone should create overwhelming concern for politicians and agencies that fluoridation may be a major cause of developmental disorders. The webpage also states mental retardation is more common for children from lower income families and for certain racial and ethnic groups. These are the same children that are targeted for fluoridation.

- Endocrine Disruption

Correlation of diagnosed hypothyroidism with fluoridation levels

“Between 4% and 5% of the U.S. population may be affected by deranged thyroid function, making it among the most prevalent of endocrine diseases.” NRC (2006) at 224-25 (citations omitted). NRC (2006) at 266 concludes that fluoride is an “endocrine disruptor.” NRC (2006) at 263 calls it a “cause for concern” that asymptomatic hypothyroidism in pregnant mothers is inversely correlated with the IQ of the offspring. A recent study in England, found a positive correlation between fluoride levels in water and hypothyroidism. Nearly 8000 areas, with about 99% of the country’s population, were studied. Areas with drinking water fluoride above 0.3 mg/L were found to be 30% more likely to have diagnosed hypothyroidism in more that 3.57 percent of the area’s population. The study was controlled for sex, age, and social-economic status in the various areas but not for iodine deficiency. Hypothyroidism leads to neuropsychiatric impairments. <http://www.endocrine-abstracts.org/ea/0011/ea0011s16.htm>

- Bones

Correlation of hip fractures for people 65+ years old with fluoridation levels

The York Review (2000) was limited to review of human epidemiological studies of water fluoridation (around 1 ppm fluoride). Over 3,200 primary studies were identified but only 9 studies met relevance criteria and measured Relative Risk (RR) of hip fracture for people 65+ years old in fluoridated areas compared to the risk in unfluoridated areas. York Review (2000) at 10, 48, and 99.) For these 9 studies, there were only 4 analyses that produced statistically significant data (i.e. RR = 1.0 was not in the 95% Confidence Interval). Each of these statistically significant analyses show an increased risk of hip fracture for those people 65+ years old living in fluoridated areas. The studies are identified in the York Review at page 48 as:

Author (Year)	Sex	Relative Risk	95% Confidence Interval
Jacqmin-Gadda (1998)	Both	2.43	(1.1, 5.3)
Danielson (1992)	Women	1.27	(1.1, 1.5)
Jacobsen (1992)	Women	1.08	(1.06, 1.10)
Jacobsen (1992)	Men	1.17	(1.13, 1.22)

A Relative Risk of 1.27 means that there is a 27% higher risk of hip fractures when living in a fluoridated area (for the 65+ year old women in the Danielson (1992) study in Utah). This is evidence that some subpopulations will have increased risk of hip fracture when their water is fluoridated at 1 mg/L. With an adequate margin of safety of 10, the MCLG for fluoride must be set lower than 0.1 mg/L. (42 USC 300g-1(b)(4)(A).) "About 300,000 Americans are hospitalized for a hip fracture every year." (Connett (2010) at page 173.) "Fracture of the hip is a major cause of morbidity and mortality [disease and death] in persons 65 years of age and older." Irish Forum (2002) at 121.

● Ethics

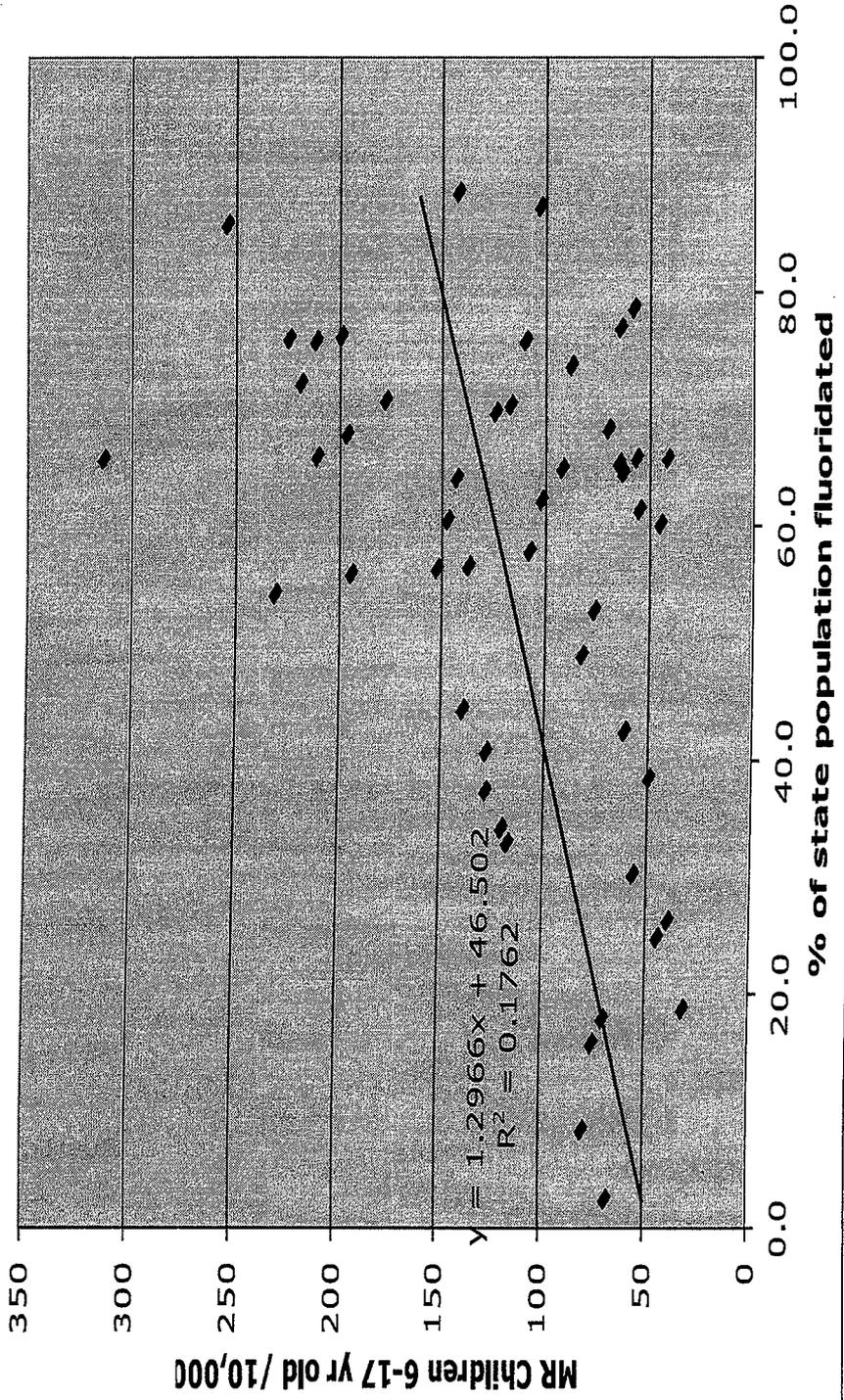
What ethical issues are of concern regarding fluoridation?

1. Should citizens be medicated with fluoridation without their consent?
2. Should fluoridation medicine be given to all to benefit a few?
3. Should fluoridation medicine be a choice so that vulnerable people are protected?
4. Should politicians who are not medical doctors be allowed to authorize treatment for their jurisdiction's whole population without consultation with each person?
5. Should public drinking water be used over the long term to deliver medicine to people?
6. Should infants and young children be given unsafe drinking water for a minimal possible benefit to older children?
7. Should people hypersensitive to fluoride be required to drink fluoridated water if they cannot afford fluoride-free water?
8. Should people be subjected to increased risks of side effects like lowered IQ in children, increased ADHD in children, increased hypothyroidism, increased hip fractures in people 65+, five- to sevenfold greater risk of contracting osteosarcoma (bone cancer) by the age of twenty for boys drinking fluoridated water when they are 6-8 years old, all for a statistically-insignificant reduction in tooth decay for older children?
9. Ethically, should a government be allowed to put a medical additive into drinking water for the benefit of the society?
10. Should the role of a water purveyor or government include medicating its customers or citizens without consultation with those customers and citizens?
11. Should water purveyors or governments be able to subject more than 42% of our children to permanent dental fluorosis by serving them fluoridated drinking water?
12. Should children with good/excellent teeth be required to ingest fluoridated water when it provides no benefit to them and only harmful side effects?
13. Should the precautionary principle be applied today because fluoridation raises threats of harm to human health? What precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically?
14. Should the Hippocratic writing *Epidemics* regarding treating disease be applied to first "do no harm"?

SUMMARY

Based on the evidence discussed above, it must be anticipated that fluoridation, even at 0.7 mg/L, will have adverse effects on the health of some persons.

**FLUORIDATION'S EFFECT ON MENTAL RETARDATION
1992**



<http://apps.nccd.cdc.gov/giscvh/map.aspx>
<http://apps.nccd.cdc.gov/nohss/FluoridationV.asp>
<http://pubs.usgs.gov/circ/2004/circ1268/htdocs/table05.html>
<http://www.cdc.gov/mmwrR/preview/mmwrhtml/00040023.htm>

Plotted by Dr. Bill Osmunson DDS

Percent of children with ADHD
versus
Percent of state population fluoridated

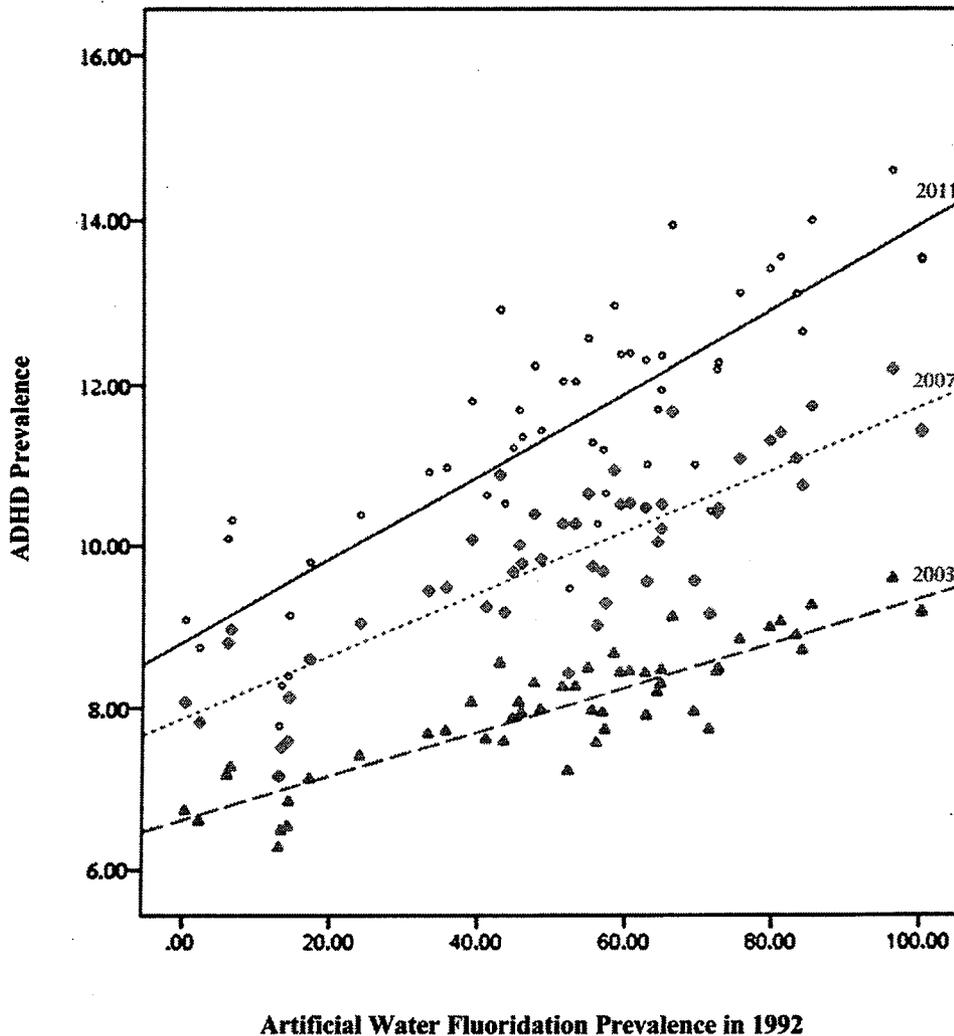


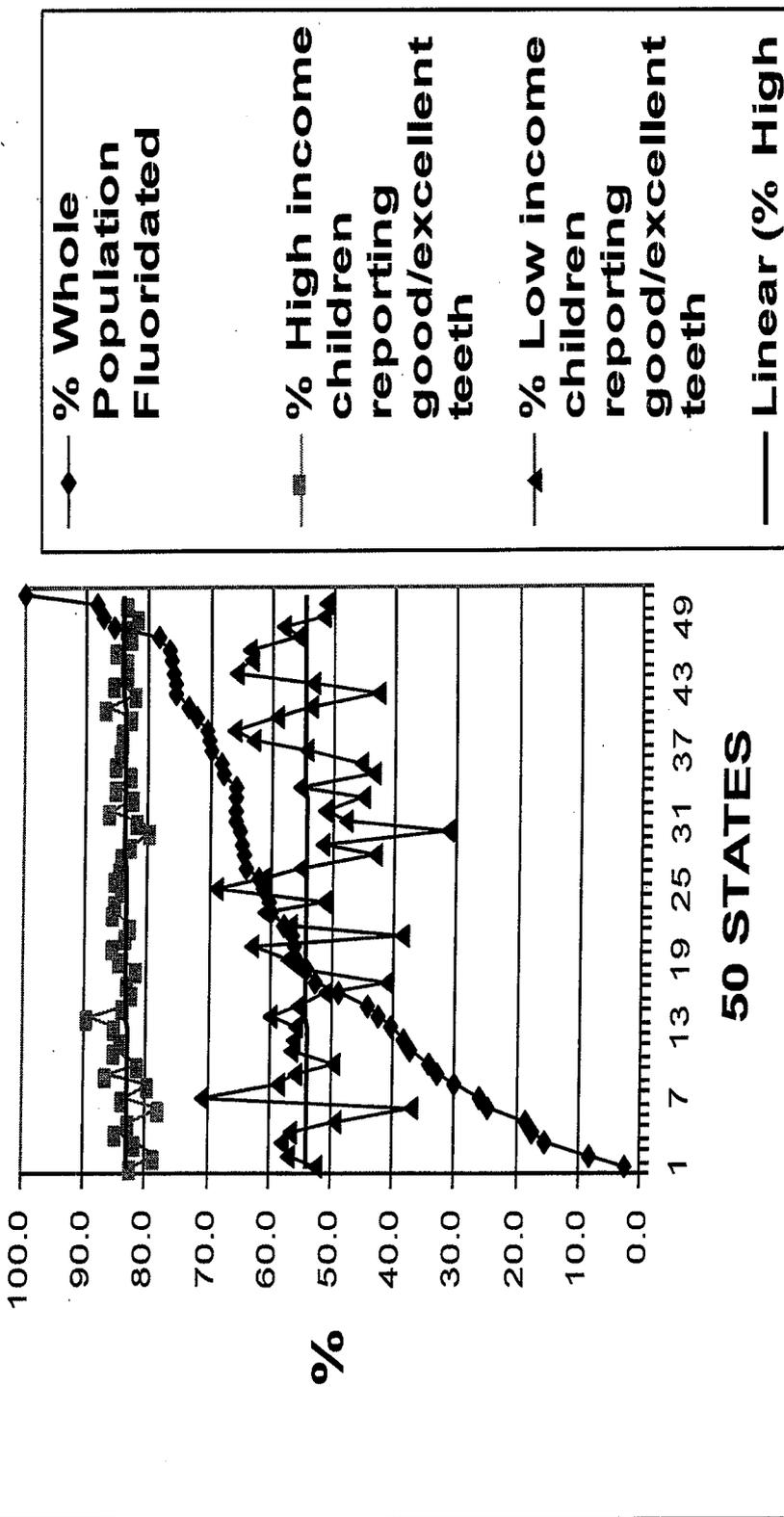
Figure 1. Artificial fluoridation prevalence predicting ADHD prevalence after adjusting for 1992 median household income, by state. Each color is for a different year of ADHD prevalence data: 2003, 2007, and 2011.

Figure and text adapted from:

Malin AJ, Till C. Exposure to fluoridated water and attention deficit hyperactivity disorder prevalence among children and adolescents in the United States: an ecological association. *Environmental Health*. 2015;14. doi:10.1186/s12940-015-0003-1. Available at: <http://www.ehjournal.net/content/14/1/17/abstract>

A-28

GOOD TEETH AND FLUORIDATION



National Survey of Children's Health. U.S. Department of Health and Human Services, Health Resources and Services Administration, Maternal and Child Health Bureau, National Survey of Children's Health 2003. Rockville, Maryland: U.S. Department of Health and Human Services, 2005

Plotted by Dr. Bill Osmunson DDS

IQ studies with water F concentration below 3 mg/L in "higher F group", and with statistically significant results

Study	IQ point difference	Water F concentration "high F group" (mg/L)
Xu et al. 1994	-14.0	1.8
Yao et al. 1997	-6.5	2
Hong et al. 2001	-6.6	2.90
Seraj et al. 2006	-13.4	2.5
Poureslami et al. 2011	-6.2	2.38

Severity of dental fluorosis on the Dean scale

of fluorosis and IQ in different groups of dental fluorosis

Group	No.	Water F	IQ	Urine F	Serum F
0	301	0.50 ± 0.53	99.76 ± 3.50	1.13 ± 0.71	0.044 ± 0.017
1	65	1.88 ± 1.07	94.18 ± 13.77	2.70 ± 1.15	0.071 ± 0.023
2	59	2.44 ± 0.66	93.27 ± 13.10	3.69 ± 1.61	0.082 ± 0.016
3	63	2.67 ± 0.63	91.51 ± 12.84	3.85 ± 1.79	0.085 ± 0.019
4	24	2.89 ± 0.81	95.33 ± 14.64	3.81 ± 1.80	0.084 ± 0.018

Xiang's presentation at FAN conference, Sept 6, 2014

3 General requirements

3.1 General

Direct additives shall be evaluated and tested in accordance with Annexes A and B. The SPAC of a contaminant shall be calculated as outlined in Annex A. Under the provisions of this Standard, a product shall not contribute any contaminant to drinking water in excess of the contaminant's SPAC.

Direct additives under this Standard shall be:

- the treatment or water supply product itself;
- the product-specific contaminants listed in each of the product sections of this Standard; and
- other constituents as identified in the formulation review.

Figure 3.1 provides an overview of the evaluation process.

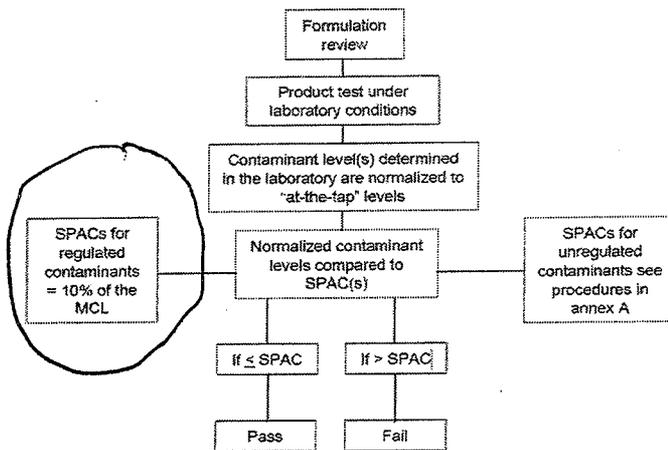


Figure 3.1 – Product evaluation overview

A.6 Risk estimation for published assessments

Calculation of the SPAC is intended to account for the potential contribution of a single substance by multiple products or materials in the drinking water treatment and distribution system. In any given drinking water treatment and distribution system, a variety of products and materials may be added to or contact the treated water prior to ingestion. The SPAC calculation is intended to ensure that the total contribution of a single substance from all potential sources in the drinking water treatment and distribution system does not exceed its acceptable concentration.

A.6.1 SPAC calculation for regulated substances

To calculate the SPAC, an estimate of the number of potential sources of the substance from all products in the drinking water treatment and distribution system shall be determined. The SPAC shall be calculated as follows:

$$\text{SPAC (mg/L)} = \frac{\text{promulgated regulatory value (mg/L)}}{\text{estimated number of drinking water sources}}$$

If available the unrounded estimated risk estimation that the promulgated regulatory value is based on shall be used in the calculation of the SPAC. In the absence of specific data regarding the number of potential sources of the substance in the drinking water treatment and distribution system, the SPAC shall be calculated as 10% of the promulgated regulatory value. The calculated SPAC shall be rounded to one significant figure, unless it is based on a regulatory value with more than one significant figure. In that case the SPAC shall be rounded to the same number of significant figures as the regulatory value.

From: [Gerald Steel](#)
To: [Phillips, Theresa \(DOH\)](#)
Cc: [Audrey Adams](#); [Scott Shock](#); [Bill Osmunson](#)
Subject: WAC 246-290-460 Rulemaking - Request to amend this regulation to require compliance with SPAC requirements in ANSI/NSF Standard 60
Date: Tuesday, February 23, 2016 12:54:25 PM
Attachments: [Attachments A-6 to A-32 to Request to Amend Regulation to comply with SPAC.pdf](#)

I submit this comment on proposed WAC 246-290-460 on behalf of myself and King County Citizens Against Fluoridation.

Under WAC 246-290-220(3) adopted by the State Board of Health, Fluorides to be added to drinking water "must comply with ANSI/NSF Standard 60." I request that WAC 246-290-460 be amended to ensure compliance with ANSI/NSF Standard 60 with respect to the SPAC (Single Product Allowable Concentration) requirements of said Standard 60. Fluorides have a unique standing in ANSI/NSF Standard 60. All chemicals, except Fluorides, certified under ANSI/NSF Standard 60 are intended to treat water to make drinking water safe (potable) and reliable (i.e. to treat the water). Fluorides, certified under ANSI/NSF Standard 60, are not needed to make drinking water safe (potable) and reliable. They are solely added to prevent and reduce tooth decay disease and the water is simply the delivery mechanism for this medication (i.e. to medicate people).

While this idea may have been historically practical 70 years ago in the medical dark ages, today it is simply entrenched and highly unethical. It is medical treatment for people without their consent, without warnings of harms, without patient checkups, and importantly, without significant effectiveness.

Regarding effectiveness, I recently reviewed the 2005 Smile Survey in Clallam County that surveyed 946 Clallam County 8 and 9 year olds for caries (treated and untreated tooth

decay). Using all of the data for 8 and 9 year olds in the survey, I found 35.6% of the students in unfluoridated areas were caries-free while only 30.2% of the students in the fluoridated areas were caries-free. Fluoridation was demonstrated by this Department of Health survey data to not be effective in Clallam County.

Under the Safe Drinking Water Act, "EPA does not have responsibility for substances added to water solely for preventative health care purposes, such as fluoride" except to address Maximum Contaminant Level (MCL) violations. (Att. A-6 to A-7 hereto.) The State Board of Health has adopted two regulations that regulate addition of Fluorides to drinking water: WAC 246-290-220(3) which requires Fluorides to comply with ANSI/NSF Standard 60; and WAC 246-290-460 which sets operational and reporting requirements when Fluorides are added to drinking water. Neither of these regulations "are related to the requirements of the Federal Safe Drinking Water Act in Washington State." (Att. A-8 hereto.)

Section 3.1 of ANSI/NSF Standard 60 (2013) states: "Direct Additives shall be evaluated and tested in accordance with Annexes A and B. The SPAC of a contaminant shall be calculated as outlined in Annex A. Under the provisions of this Standard, a product shall not contribute any contaminant to drinking water in excess of the contaminant's SPAC." (A-32 hereto.) Direct Additives include the product itself and other contaminants. (*Id.*) The SPAC (Single Product Allowable Concentration) is "The maximum concentration of a contaminant in drinking water that a single product is allowed to contribute under Annex A of this Standard." (Sec. 2.25 (section numbers refer to sections of ANSI/NSF Standard

60 (2013)).) A contaminant is "Any physical, chemical, biological, or radiological substance in Water" which may have a beneficial or detrimental effect on the potability of water. (Sec. 2.9.) The maximum contaminant level (MCL) is "The maximum concentration of a contaminant permitted in a public drinking water supply as defined by the federal Safe Drinking Water Act." (Sec. 2.18.) As you likely know the MCL for Fluorides is 4.0 mg/l in the federal Safe Drinking Water Act. (40 CFR 141.62 (A-22 hereto).)

Fluorides are considered contaminants to drinking water under ANSI/NSF Standard 60 even if they are added to prevent and reduce tooth decay disease. When all of the sources of contamination in drinking water are not specifically analyzed, the standard SPAC for Fluoride contaminants is 10% of the MCL. (Sec. A.6.1 and Figure 3.1 on page 6 of the Standard (A-32 hereto).) Therefore, using this 10%, the standard SPAC or maximum Fluoride that fluoridation chemicals can add to drinking water is 10% of the 4.0 MCL or 0.4 mg/l.

WAC 246-290-460 that governs implementation of fluoridation in Washington State fails to address SPAC requirements for Fluorides. While there is an alternative calculation of SPAC provided in Sec. A.6.1 (A-32 hereto), this calculation requires collection and analysis "of specific data regarding the number of potential sources of [Fluorides] in the drinking water treatment and distribution system." To the best of my knowledge, no purveyors of fluoridated public water use this alternative calculation of SPAC.

So if every purveyor of fluoridated public water is using the standard SPAC to determine the maximum Fluoride it can add to its water supply, then every one of these purveyors is

adding Fluoride to its drinking water in a manner that does not comply with ANSI/NSF Standard 60. This is because every such purveyor is adding more than the allowed standard SPAC of 0.4 mg/l of Fluoride to its water supply. Proposed WAC 246-290-460(2) does not allow a purveyor to fluoridate unless the Fluoride level after fluoridation is at least 0.5 mg/l. The 2012 NSF Fact Sheet on Fluoridation at page 3 states that, "The data-derived SPAC for the fluoride ion in drinking water from NSF certified treatment products is 1.2 mg/L, or less than one-third of the EPA's MCL." This statement is not part of ANSI/NSF Standard 60 (see Standard 60 at iii) and is generally erroneous. This is actually the maximum amount of fluoride ion (or fluorine) in drinking water that was authorized in 1962 by the U.S. Public Health Service. It is not in compliance with the standard SPAC allowed by ANSI/NSF Standard 60 and NSF cannot know what the calculated SPAC is for individual water purveyors. We request that you amend WAC 246-290-460 to require data-derived SPAC to be reported to the State Department of Health when fluoridation chemicals will add 0.4 mg/l or more fluoride to Group A public drinking water. This information is required to show compliance with the SPAC requirements of ANSI/NSF Standard 60 and compliance with ANSI/NSF Standard 60 is required by WAC 246-290-220(3) for fluoridation chemicals to be added to Group A public drinking waters.

A-21 hereto is a letter from the State Department of Health refusing to determine if the certified ANSI/NSF Standard 60 Fluorides that it regulates actually comply with ANSI/NSF Standard 60. This must be resolved.

New data from more than 100 animal studies and more than 50

human studies demonstrate that Fluorides are neurotoxic. Fluorides cause lowered IQs in offspring when pregnant mother's drink fluoridated water and when infants are fed formula made with fluoridated water. (*See* A-23 to A-31, a paper I wrote for NIEHS/NTP for a teleconference with the Director of those agencies and with others earlier this year.)

Gerald Steel

Attorney at Law

7303 Young Rd. NW

Olympia WA 98502

Tel/Fax (360) 867-1166

New studies cast doubt on fluoridation benefits

An analysis of national survey data collected by the National Institute of Dental Research (NIDR) concludes that children who live in areas of the U.S. where the water supplies are fluoridated have tooth decay rates nearly identical with those who live in nonfluoridated areas.

The analysis was done by John A. Yiamouyiannis, a biochemist and expert on the biological effects of fluoride, who has been an ardent opponent of fluoridation for 20 years. His results are not widely different from those recently found—but as yet unpublished—by NIDR in analyzing the same data.

In the 1986-87 school year, NIDR examiners looked for dental caries in 39,207 schoolchildren aged five to 17 from 84 different geographical areas. Yiamouyiannis obtained the survey data from NIDR under the Freedom of Information Act.

Yiamouyiannis compared decay rates in terms of decayed, missing, and filled permanent teeth. The average decay rates for all the children aged five to 17 were 2.0 teeth for both fluoridated and nonfluoridated areas. When he omitted those children who had ever changed addresses, and thus confined the study to children with an unchanging fluoridation status, the results were nearly the same—a decay rate of 2.0 for fluoridated areas, and 2.1 for nonfluoridated areas. Decay rates in the individual age groups were sometimes lower in fluoridated areas, sometimes lower in nonfluoridated areas. The differences were never greater than 0.5 teeth. He has submitted his study for publication in the Danish journal *Community Dentistry & Oral Epidemiology*.

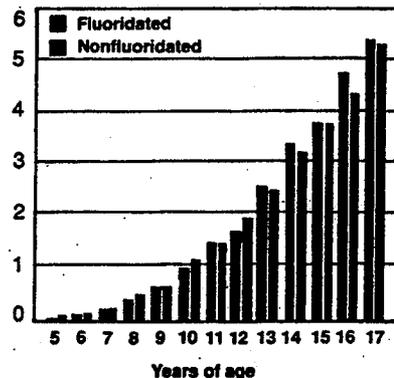
He also found that the percentages of decay-free children were virtually the same in fluoridated and nonfluoridated areas, and averaged about 34%. This analysis included

both permanent and deciduous (baby) teeth. NIDR's claim that 50% of the children in the U.S. are decay-free, headlined in newspapers across the country last summer, was based largely on the fact that NIDR analyzed only permanent teeth in children aged five to 17, and a large fraction of these children were not old enough to have many permanent teeth, Yiamouyiannis says.

When analyzing the survey data,

Tooth decay rates appear unrelated to fluoridation

Average decayed, missing, and filled permanent teeth per child



Note: Averages are for the U.S. only. Areas where the fluoridation status was mixed or changed at some time since 1970 have been omitted. Source: National Institute of Dental Research data analyzed by John Yiamouyiannis

NIDR compared decay rates in two ways: in terms of the number of decayed, missing, and filled permanent teeth; and in terms of decayed, missing, and filled surfaces of teeth. Both of these methods are widely used today. NIDR found that children who have always lived in fluoridated areas have 18% fewer decayed surfaces than those who have never lived in fluoridated areas. But when NIDR analyzed the data in terms of teeth, the differences were smaller. Janet A. Brunelle, statistician in the epidemiology program at NIDR, tells C&EN the results for teeth "are in a box somewhere" and she does not remember exactly what they are.

Brunelle says NIDR is publishing only the results for surfaces because they are more meaningful. Surface

rates give a more complete picture of the extent of decay, she adds, and the decay rate for teeth "is rather low so that there is very little difference in most anything." When asked to comment on Yiamouyiannis' results, Brunelle said she didn't know whether they are valid.

In reaction to Yiamouyiannis' new study, the union of professional employees at the Environmental Protection Agency has written a letter to EPA Administrator William K. Reilly. The letter asks him to "immediately suspend (not revoke) EPA's unqualified support for fluoridation" until the agency conducts its own assessment of the risks and benefits of fluoride exposure. The union, Local 2050 of the National Federation of Federal Employees, has been concerned for some time that EPA evaluated fluoride politically, rather than scientifically. The union also believes the safe level of fluoride in drinking water should have been lowered rather than raised in 1986, when EPA increased the maximum allowable contaminant level to 4 ppm from a range of 1.4 to 2.4 ppm.

Another analysis of decay rates is published in the current issue of the *American Journal of Public Health*. Jayanth V. Kumar of the New York State Department of Health examined decay rates in seven to 14 year olds in Newburgh, N.Y., which has been fluoridated since 1945, and in nearby Kingston, which has never been fluoridated. He found that the caries prevalence in Newburgh—1.5 decayed, missing, and filled permanent teeth—is somewhat lower than it is in Kingston (2.0). However, since the 1954-55 school year, the decay rate has actually declined more in nonfluoridated Kingston than in Newburgh.

When asked by C&EN, a spokesman for the American Dental Association said that ADA believes that water fluoridation can reduce tooth decay 18 to 25%. But as recently as 1988 the association claimed fluoridation reduces decay 40 to 60%.

Bette Hileman

**FLUORIDE IN DRINKING WATER:
A Scientific Review of EPA's Standards**

Committee on Fluoride in Drinking Water
Board on Environmental Studies and Toxicology
Division on Earth and Life Studies

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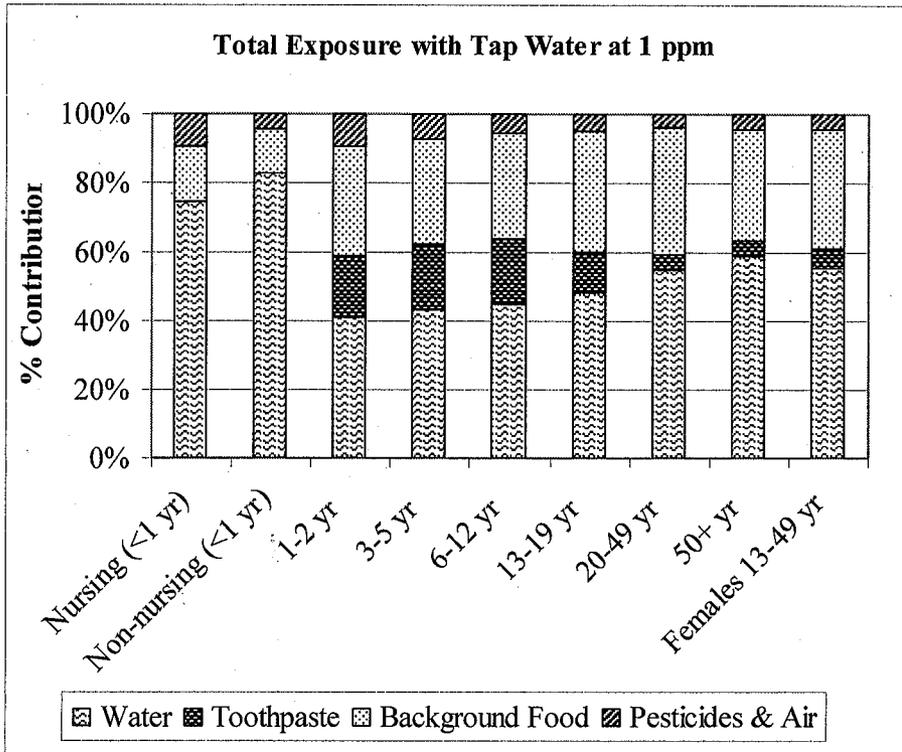
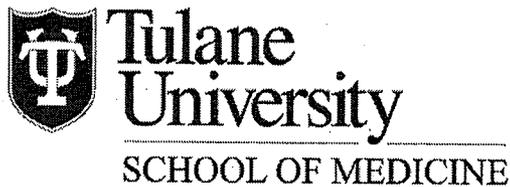


FIGURE 2-1 Source contribution to total inorganic fluoride exposure, including fluoride at 1 mg/L in tap water. The estimated chronic inorganic fluoride exposures from the various routes are presented in Tables 2-9 and 2-10. No fluoride supplement is included for any population subgroup. The total exposures as presented in Table 2-11 for the population subgroups are: 0.030 mg/kg/day (nursing infants), 0.087 mg/kg/day (non-nursing infants), 0.066 mg/kg/day (1-2 years old), 0.060 mg/kg/day (3-5 years old), 0.040 mg/kg/day (6-12 years old), 0.028 mg/kg/day (13-19 years old), and 0.031 mg/kg/day for adults (20 to 50+ years old) and women of child-bearing age (13-49 years old).

toothpaste, children inappropriately given fluoride supplements in a fluoridated area, children in an area with high fluoride concentrations in soil, and children with pica who consume large amounts of soil.

The exposure estimates presented in this chapter for non-drinking water routes are based on the potential profile of fluoride residue concentrations in the current exposure media. They likely do not reflect the concentration of past exposure scenarios, particularly for routes that show changes in time (e.g., pesticide use practices). Any new and significant source of fluoride exposure, such as commodities approved for sulfuryl fluoride fumigation application beyond April 2005, is expected to alter the percentage of drinking water contribution as presented in this chapter.

Different assumptions for the drinking water concentration alone also can result in slightly different estimates. For example, values in Table 2-11 are derived from assuming that the nontap water has a fixed fluoride concentration of 0.5 mg/L, while tap water concentration varies up to 4 mg/L. Table 2-12 provides alternative calculations of total exposure by assuming that all sources of drinking water (both tap and nontap water) contain the same specified fluoride



Date: May 31, 2012

Gerald Steel PE
Attorney at Law
7303 Young Road NW
Olympia WA 98502
geraldsteel@yahoo.com

I am qualified as an expert in fluoridation by knowledge, skill, experience, training, or education and in my opinion, the bulk fluoridation products, fluorosilicic acid, sodium fluorosilicate, and sodium fluoride, are not "safe and effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water at 0.7 to 1.3 ppm of fluoride ion.

Signed:

A handwritten signature in black ink that reads "Howard W. Mielke". The signature is written in a cursive style.

Dr. Howard W. Mielke, Ph.D.
Toxicologist, Department of Pharmacology

Credentials:

I conducted a major empirical study that included discussion about the synergistic impact of lead and fluoride on learning among the children of New Orleans.

Citation:

S. Zahran, H.W. Mielke, S. Weiler, K.J. Berry, C. Gonzales. 2009. Children's blood lead and standardized test performance response as indicators of neurotoxicity in metropolitan New Orleans elementary schools. *NeuroToxicology* 30:888-897.
<http://dx.doi.org/10.1016/j.neuro2009.07.017>

Gerald Steel

From: Bill Osmunson [bill@teachingsmiles.com]
Sent: Friday, June 01, 2012 8:00 AM
To: 'Gerald Steel'
Subject: Signed statement as qualified expert

I am qualified as an expert in fluoridation by knowledge with over 11,000 hours devoted to the study of fluoride used for dental purposes, skill, experience, training in teaching the public and health care professionals regarding fluoride, education with degrees as a dentist and Masters Degree in Public Health and in my opinion, the bulk fluoridation products, fluorosilicic acid, sodium fluorosilicate, and sodium fluoride, when diluted in water are not "safe or effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water above 0.02 ppm of fluoride ion.

The most precise statement which is fully supported by science, law, and ethics is by the EPA professionals through their union:

"In summary, we hold that fluoridation is an unreasonable risk. That is, the toxicity of fluoride is so great and the purported benefits associated with it are so small - if there are any at all – that requiring every man, woman and child in America to ingest it borders on criminal behavior on the part of governments."

- ***Dr. J. William Hirzy, Senior Vice-President, Headquarters Union,***
- ***US Environmental Protection Agency, March 26, 2001***

Signed

Bill Osmunson DDS, MPH
25977 Canyon Creek #G
Wilsonville, OR 97070
bill@teachingsmiles.com

Date: June 1, 2012

Gerald Steel

From: spittle@ihug.co.nz
Sent: Wednesday, May 30, 2012 1:16 PM
To: Gerald Steel
Subject: Fluoridation chemicals

727 Brighton Road
Ocean View
Dunedin 9035
New Zealand

Phone / Fax +64 3 4811418

To whom it may concern

I am qualified as an expert in fluoridation by knowledge, skill, experience, training, or education and in my opinion, the bulk fluoridation products, fluorosilicic acid, sodium fluorosilicate, and sodium fluoride, are not "safe and effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water at 0.7 to 1.3 ppm of fluoride ion.

I have studied fluoridation since 1988, published on the effects of fluoride including a book Fluoride Fatigue, been a peer reviewer for the 2000 University of York review of fluoridation, and, after serving for some years as an Associate Editor, been Managing Editor of the journal Fluoride since 1999. My publications have been referred to in both the University of York review, 2000, and the NRC review of 2006.

Signed: Bruce Spittle MB ChB DPM (Otago) FRANZCP

Date: 31 May 2012

Gerald Steel

From: David Kennedy [davidkennedy-dds@cox.net]
Sent: Wednesday, May 30, 2012 12:41 PM
To: geraldsteel@yahoo.com
Cc: Tara Blank, Ph.D.
Subject: Fluoride as a drug
Attachments: NRC-2006.pdf; ATT00523.htm; KMT.MWD.remarksFinal.pdf; ATT00526.htm; Opflow.pdf; ATT00529.htm

I am qualified as an expert in fluoridation by knowledge, skill, experience, training, and education and in my opinion, the bulk fluoridation products, fluorosilicic acid, sodium fluorosilicate, and sodium fluoride, are not "safe and effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water at 0.7 to 1.3 ppm of fluoride ion.

My opinion in this matter has evolved over the course of my career as the scientific evidence on systemic exposure to fluoride and theoretical mechanisms of action have evolved. I am the past president of the International Academy of Oral Medicine and Toxicology and a lifetime member since its inception, the first organization to fund research, perform risk assessment and bring scientific methodologies to evaluate the safety of materials used in the evidence based practice of dentistry.

Today it is well recognized that any beneficial impact that fluoride may have upon tooth decay is likely entirely topical at levels 1000 times higher than those achieved by fluoridation. (Featherstone JADA 2000) Furthermore fluoride incorporated into the enamel and tooth systemically has no measurable impact on acid solubility (tooth decay) op cit. Since advocates for fluoride use now acknowledge that the effects are topical there is no conceivable benefit for systemic exposure to this element. There are however well documented adverse effects from even minimal systemic fluoride exposures especially in vulnerable human subsets such as kidney patients or infants. (NRC 2006 Note:Table 8-2) summarized by Dr. Thiessen for the Metropolitan Water District. attached:

**FLUORIDE IN DRINKING WATER:
A Scientific Review of EPA's Standards**

Committee on Fluoride in Drinking Water
Board on Environmental Studies and Toxicology
Division on Earth and Life Studies

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TABLE 8-2 Summary of Major Observed Endocrine Effects of Fluoride in Humans, with Typical Associated Intakes and Physiological Fluoride Concentrations

End Point	Fluoride Intake, mg/kg/day ^a	Fluoride in Serum or Plasma, mg/L	Fluoride in Urine, mg/L	Key References
Altered thyroid function (altered T4 and/or T3 concentrations)	0.05-0.1 (0.03 with iodine deficiency)	≥0.25 ^a	2.4	Bachinskii et al. 1985; Lin et al. 1991; Yang et al. 1994; Michael et al. 1996; Susheela et al. 2005
Elevated TSH concentrations	0.05-0.1 (0.03 with iodine deficiency)	≥0.25 ^a	≥2	Bachinskii et al. 1985; Lin et al. 1991; Yang et al. 1994; Susheela et al. 2005
Elevated calcitonin concentrations	0.06-0.87	0.11-0.26 ^b	2.2-18.5 mg/day	Teotia et al. 1978
Goiter prevalence ≥ 20%	0.07-0.13 (≥ 0.01 with iodine deficiency)	NA ^c	NA	Day and Powell-Jackson 1972; Desai et al. 1993; Jooste et al. 1999
Impaired glucose tolerance in some individuals	0.07-0.4	0.08 ^a 0.1-0.3 ^b	2-8	Rigalli et al. 1990, 1995; Trivedi et al. 1993; de la Sota 1997
Increased parathyroid hormone concentrations, secondary hyperparathyroidism, in some individuals	0.15-0.87	0.14-0.45 ^b	3-18.5 mg/day	Juncos and Donadio 1972; Teotia and Teotia 1973; Larsen et al. 1978; Teotia et al. 1978; Duursma et al. 1987; Dandona et al. 1988; Stamp et al. 1988, 1990; Pettifor et al. 1989; Srivastava et al. 1989; Dure-Smith et al. 1996; Gupta et al. 2001

^aSerum.

^bPlasma.

^cNot available.

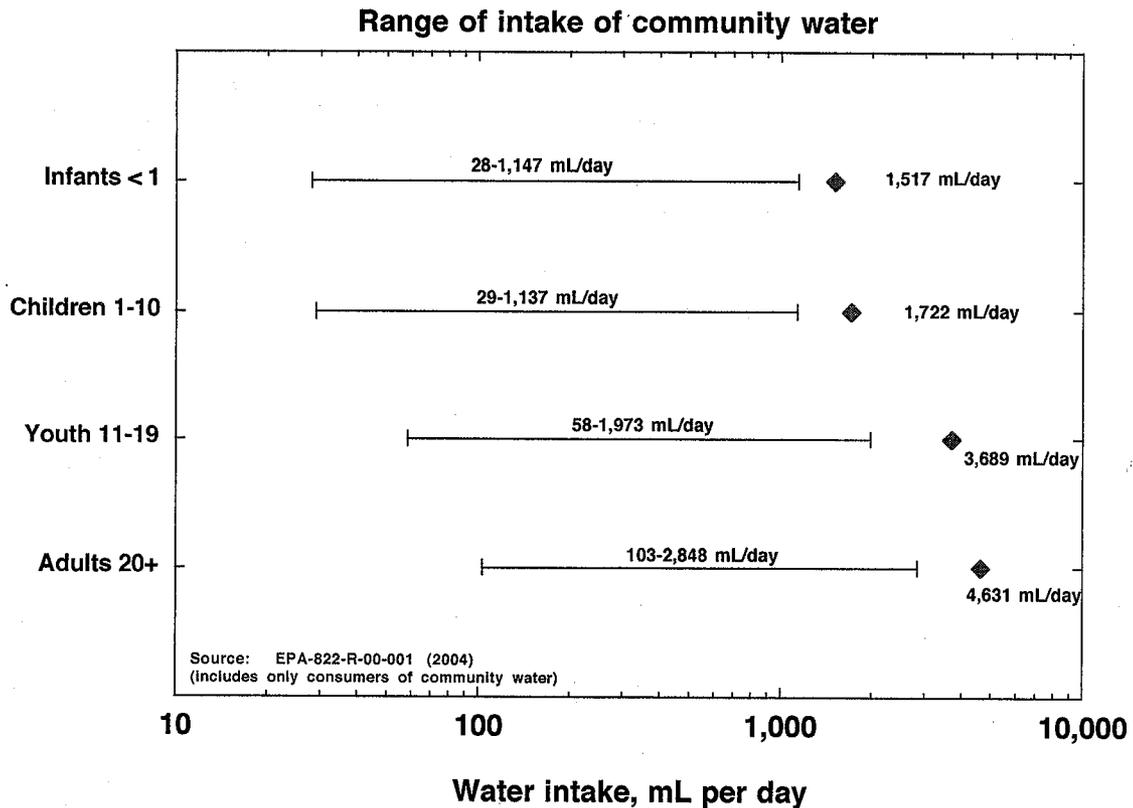
Adverse Health Effects from Fluoride in Drinking Water

Comments to the Water Quality and Operations Committee
Metropolitan Water District
Los Angeles, California
August 20, 2007

Kathleen M. Thiessen, Ph.D.
SENES Oak Ridge, Inc.
Center for Risk Analysis
102 Donner Drive
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(865) 483-6111
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Thank you for this opportunity to address the committee. I understand that your plans to fluoridate are already in place. I wish simply to inform you of some of the implications of those plans.

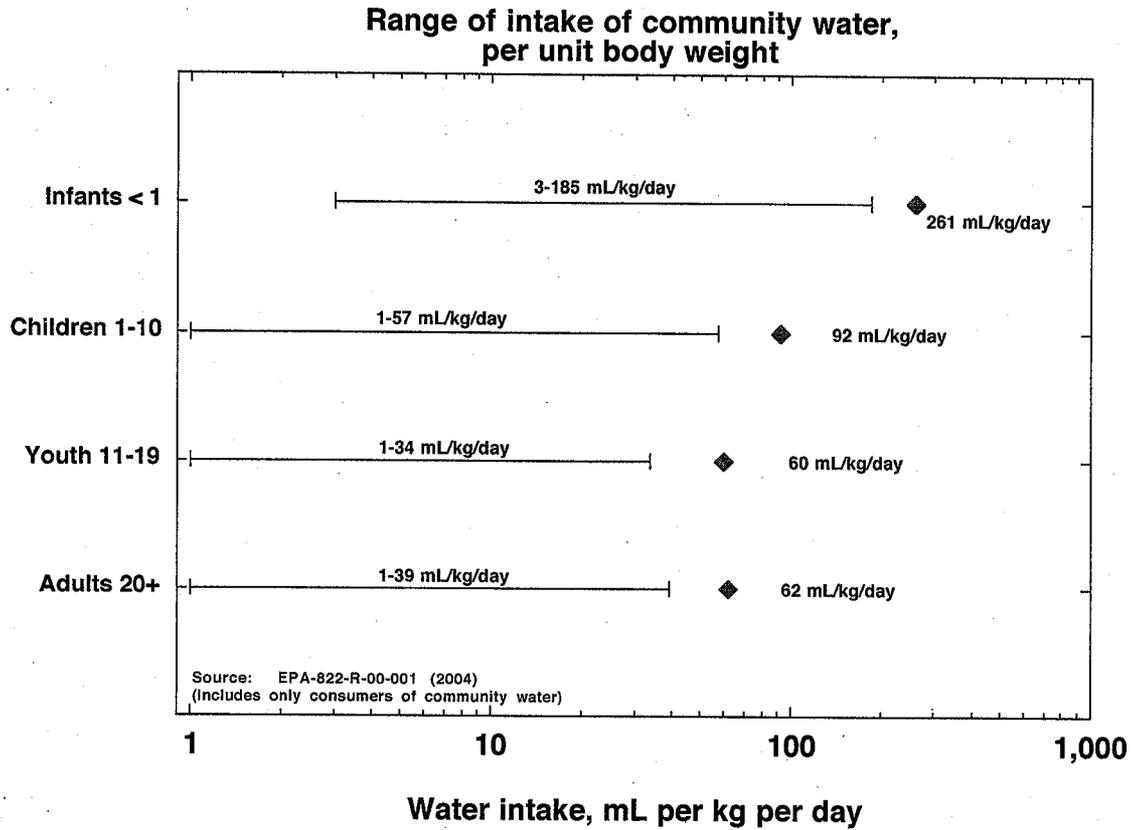
Slide 1



The first graph illustrates the expected range of consumption of community water (public tap water) for various age groups, in quantities of milliliters per day (mL per day). The ranges include only people who actually consume tap water. Note that some people consume substantially more tap water than the usual range (indicated by the diamonds). This information is from an EPA report published in 2004.

The total consumption of community water shown here is not to be confused with total fluid consumption or total water consumption. It does not include well water, bottled water, or commercial beverages. It does include water consumed directly and water used to prepare household or restaurant foods and beverages.

Slide 2

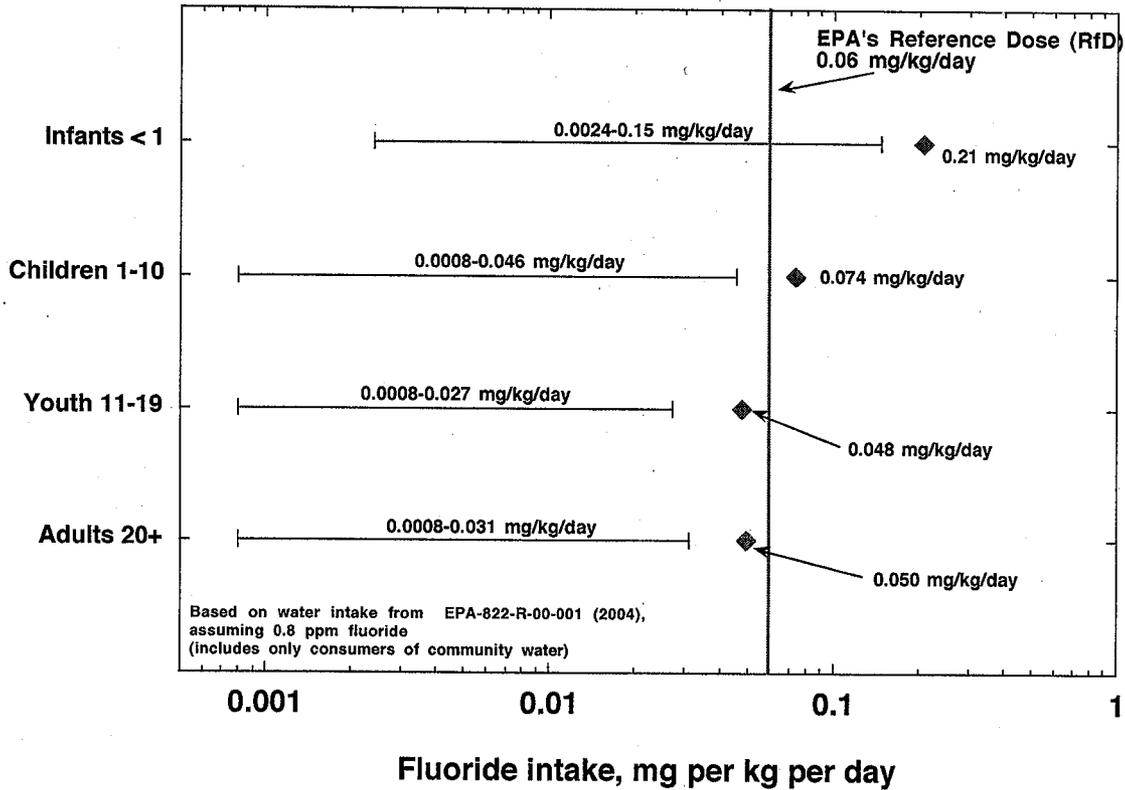


The second graph shows the same information as in the first slide, but in terms of water intake per unit body weight (milliliters of community water intake per kg of body weight, or mL per kg per day). Note that infants have the highest tap water consumption per unit body weight, with some infants reaching more than 250 mL per kg per day.

In general, the people with the highest tap water intakes include babies fed formula made with tap water, people with certain medical conditions (e.g., diabetes insipidus, diabetes mellitus) or taking certain medications (e.g., lithium), people in unairconditioned residences in hot climates, people who work outside in hot climates or do heavy physical labor, and athletes.

Slide 3

Range of fluoride intake from community water, assuming 0.8 ppm fluoride in the water

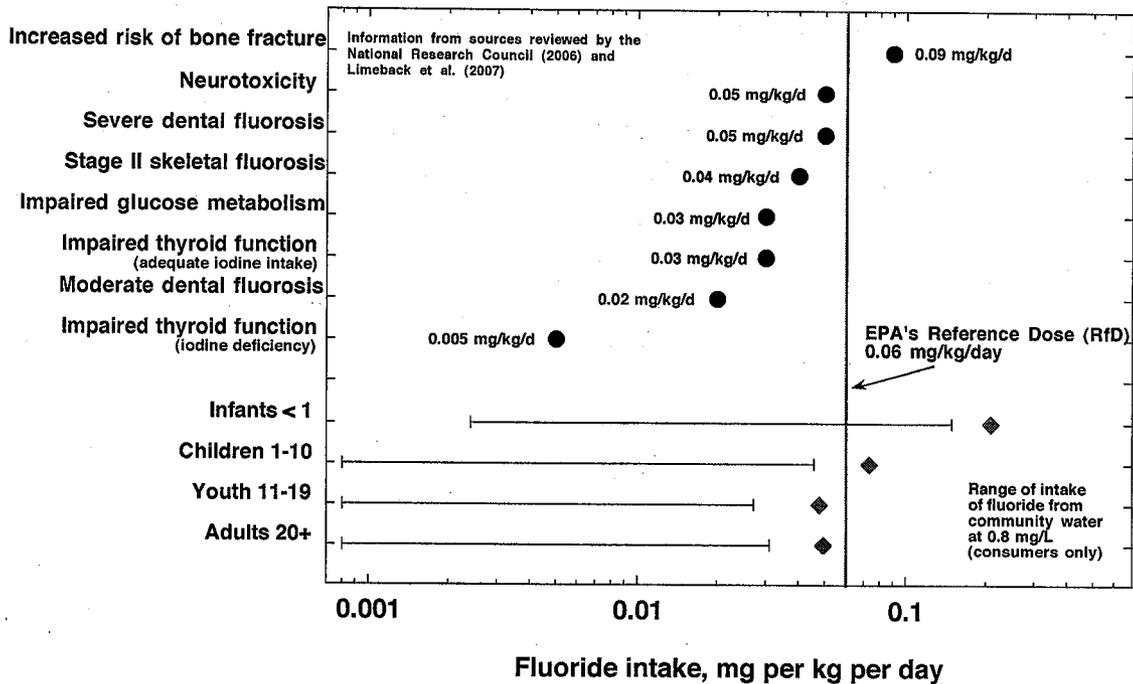


The third graph shows estimated fluoride intakes for each age group (mg of fluoride per kg of body weight per day), assuming the range of tap water intakes shown in Slide 2 and a fluoride concentration in the tap water of 0.8 ppm (0.8 mg fluoride per liter of water). Also shown is EPA's reference dose, which is defined as "an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." For fluoride, the reference dose is 0.06 mg per kg per day. As seen in the graph, many infants have a fluoride intake just from tap water that exceeds EPA's reference dose for fluoride. Children (ages 1-10) with high water consumption also exceed EPA's reference dose. Older children (youth) and adults with high water consumption are very close to EPA's reference dose.

Note that this graph shows estimated fluoride intakes only from tap water. These estimates do not include fluoride intakes from other sources, such as commercial beverages (which are often made with fluoridated tap water), toothpaste, tea, or food. When these other sources of fluoride intake are included, total fluoride intakes for many members of all age groups exceed EPA's reference dose.

Slide 4

Estimated "No-effect" levels in humans



The final graph shows the estimated fluoride intakes from tap water from Slide 3, plus estimates of the "no-effect" levels for various adverse health effects. These "no-effect" levels represent fluoride intakes at or below which most people are not expected to experience any harmful effects. Note that these estimates are based on average exposures of study populations; these estimates do not include any margin of safety, and they might not be protective for all individuals. Intakes above these levels cannot be considered safe.

Note also that most of these "no-effect" levels are lower than EPA's reference dose for fluoride. In other words, EPA's reference dose is not protective for most of these health endpoints.

Note also that most of these "no-effect" levels are exceeded by many members of the population, of all ages, just from fluoride at 0.8 ppm in community drinking water. When other fluoride sources are included, even more people are expected to exceed the "no-effect" levels. In order to be "safe" for all members of the population, fluoride intakes for all people must be kept below the lowest "no-effect" levels, when all sources of fluoride intake are included, and with an adequate margin of safety.

This list of adverse health effects does not include cancer. A carcinogenic (cancer-causing) effect of fluoride cannot be ruled out from the available data, and at the very least, a cancer-promoting effect is likely. For carcinogenic substances, the risk of cancer increases with the amount of exposure, such that even a very low exposure carries with it some cancer risk.

In conclusion, I would like to quote from the Director of Laboratories, Department of Water Supply, Gas and Electric, of the City of New York, from a presentation made in 1956 but still relevant today:

The continued promotion of water supply fluoridation in [the] face of mounting adverse evidence and criticism requires some evaluation. It seems that the proponents hit upon an idea years ago which appealed to them, and which they felt was sound. As their claims for safety were progressively discredited, rather than acknowledge this, they persisted in condoning such evidence. At the same time they were lending their prestige to such equivocation. Certainly the proponents of fluoridation are not intent upon poisoning or harming anyone, however, the dilemma of prestige is a very difficult matter to resolve.

The proponents have tried to demonstrate various factors of safety which are patently naïve. . . . It has been customary to consider a minimal factor of safety of not less than 10 for substances which may be admitted to water supplies. This would mean that ten times the amount of the proposed substance when present in the water supply would be definitely without harm to human or beast. It is obvious from the knowledge of fluoride toxicity that such factor of safety cannot be established when fluoride is added to the public water supply at the level recommended by the proponents of fluoridation. In view of the fact that the range of water consumption may vary over a ratio of 20 to 1 the insistence upon a factor of safety of 10 is exceedingly moderate.

It must be concluded that the fluoridation of public water supplies is a hazardous procedure, people are bound to get hurt, it remains to find out how many and when. I do not believe the water supply fraternity is interested in demonstrating this with wholesale experimentation on populations.

Thank you.

References

NRC (National Research Council). 2006. Fluoride in Drinking Water: A Scientific Review of EPA's Standards. Washington, DC: The National Academies Press. [Available at <http://www.nap.edu/catalog/11571.html>]

Limeback, H., Thiessen, K.M., Isaacson, R.L., and Hirzy, W. 2007. The EPA MCLG for fluoride in drinking water: New recommendations. Presentation to the Society of Toxicology 2007 Annual Meeting. *Toxicological Sciences* 96(1):317.

Nesin, B.C. 1956. A water supply perspective of the fluoridation discussion. *J. Maine Water Util. Assoc.* 32:33-47.

U.S. Environmental Protection Agency. 1989. Fluorine (soluble fluoride) (CASRN 7782-41-4). Integrated Risk Information System. [Available at <http://www.epa.gov/iris/subst/0053.htm>]

U.S. Environmental Protection Agency. 2004. Estimated Per Capita Water Ingestion and Body Weight in the United States—An Update Based on Data Collected by the United States Department of Agriculture's 1994–1996 and 1998 Continuing Survey of Food Intakes by Individuals. Washington, DC: U.S. Environmental Protection Agency, Office of Water, Office of Science and Technology, EPA-822-R-00-001.

Treatment Chemicals Contribute to Arsenic Levels

By Cheng-nan Weng, Darrell B. Smith,
And Gary M. Huntley

Arsenic is an issue that water utilities no longer can avoid. The US Environmental Protection Agency is expected to propose a reduction in the federal drinking water standard on arsenic from 50 µg/L to 5 µg/L later this year, although USEPA is also considering setting the maximum contaminant level at 3 µg/L, 10 µg/L, and 20 µg/L. The final arsenic rule is due by Jan. 1, 2001.

Utilities should test their sources of water for arsenic and compare them with the proposed levels of 3, 5, and 10 µg/L. However, testing source water alone may not be sufficient to determine the arsenic load in finished water. Some treatment chemicals may also contain trace amounts of arsenic. Utilities should review and estimate the maximum possible arsenic concentrations contributed by the chemicals they use in drinking water treatment. Even trace amounts add up and may contribute a substantial portion—possibly up to 10 percent—of a 3 or 5 µg/L maximum contaminant level.

Connecticut Experience

The South Central Connecticut Regional Water Authority has three surface water treatment plants (SWTPs) and five wellfields. Recently, SCCRWA calculated the arsenic burden derived from chemicals routinely used to treat surface and groundwater at these facilities. Those chemicals are listed in Table 1.

To estimate the trace arsenic levels in the bulk treatment chemicals, data from the suppliers' analysis report or product specifications were used. The resulting trace arsenic concentrations in the finished water that were contributed by the treatment chemicals were computed by one of the following two methods:

1. For those chemicals with dosages expressed as mg/L of product chemicals (such as polymer, sulfuric acid, bimetallic zinc metaphosphate, and potassium permanganate), the resulting trace arsenic concentration in the finished water was computed by multiplying the chemical dosage by the trace arsenic level in the bulk treatment chemical.

2. For other chemicals (such as alum, ferric chloride, caustic soda, and fluorosilicic acid), a dilution factor was determined by dividing the chemical concentration by the chemical dosage. The resulting trace arsenic concentration in the finished water was computed by dividing the trace arsenic level in the bulk treatment chemical by the dilution factor.

Information produced by several calculations is tabulated as follows:

- Table 2 shows the maximum possible arsenic concentrations contributed by treatment chemicals for one surface water treatment plant that uses alum (0.279 µg/L arsenic contributed).
- Table 3 shows the maximum possible arsenic concentrations contributed by treatment chemicals for the wellfield, which uses sodium hypochlorite for disinfection (0.249 µg/L arsenic contributed).

Treatment Chemical	# Surface Water Treatment Plants (3 total)	# Groundwater Treatment Facilities (5 total)
Sodium hydroxide	3	Not used
Sulfuric acid	1	Not used
Alum	2	Not used
Potassium permanganate	2	Not used
Ferric chloride	1	Not used
Synthetic polymer A	1	Not used
Synthetic polymer B	1	Not used
Chlorine	3	4
Sodium hypochlorite	Not used	1
Bimetallic zinc metaphosphate	3	5
Fluorosilicic acid	3	5

Table 1. Chemicals routinely used by the South Central Connecticut Regional Water Authority, and the number of facilities where they are used.

- Table 4 shows the range of maximum arsenic contribution by treatment chemicals for the SCCRWA (range of all compounds, 0.0002-0.245 µg/L).
- Table 5 compares in finished water the calculated amount of arsenic that is contributed by treatment chemicals with the analytical result (overall calculated range, 0.248—0.306 µg/L; analytical result <1µg/L in all cases).

These data show that in finished water the theoretical arsenic concentrations attributable to normal dosages of water treatment chemicals are extremely low (Tables 2, 3, and 4). This conclusion is supported by the analytical data (Table 5), which show arsenic concentrations to be below 1.0 µg/L in all of the SCCRWA's surface and groundwater-treatment facility finished waters.

Conclusion

If the standard were set at 3 µg/L, about 10 percent of the MCL would come from the treatment chemicals, hardly a minimal amount. It is also interesting to note that about 90 percent of the arsenic that would be contributed by treatment chemicals is attributable to fluoride addition.

If your processes include the addition of chemicals, ask your manufacturer for the amount of arsenic in each. If necessary, obtain conversion charts for diluted products, as well. Then calculate how much arsenic those chemicals will add to your finished water. If the total is close to the MCLs proposed by USEPA, you have reason for concern.

To find out more about the proposed arsenic rule, go to the agency's Web site, <www.epa.gov/safewater/arsenic.html>, or call the Safe Drinking Water Hotline at (800) 426-2791.

- *Cheng-nan "Mike" Weng, PhD, DEE, is senior water quality engineer; Darrell B. Smith is vice president of water quality and research, and Gary M. Huntley is water treatment manager for South Central Connecticut Regional Water Authority, 90 Sargent Drive, New Haven, CT 06511; (203) 624-6671.*

Table 2. Arsenic contributed by chemicals used to treat surface water at Lake Gaillard Water Treatment Plant

Treatment Chemical	Amount of Arsenic in Product	Dosage	Calculation of Contribution	Arsenic Contribution
50% alum	0.25 mg/L	10 mg/L*	Chemical concentration of 50% alum = 650 mg/mL Dilution factor = $650 \times 1,000 \div 10 = 65,000$ Arsenic contribution = $0.25 \div 65,000$ mg/L	0.00385 µg/L
Polymer A.	< 0.5 mg/L	2.0 mg/L	Arsenic contribution = 0.5 mg/L x 2 mg/L	0.001 µg/L
50% Sodium hydroxide (NaOH)	1.5 mg/L (maximum)	12.5 mg/L* (maximum)	Chemical concentration of 50% NaOH = 770 mg/mL Dilution factor = $(770 \times 1,000) \div 12.5 = 61,600$ Arsenic contribution = $1.5 \div 61,600$ mg/L	0.024 mg/L
Fluorosilicic acid (H ₂ SiF ₆)	Maximum = 60 mg/L Normal = 28 mg/L	1.0 mg/L* as F	H ₂ SiF ₆ solution contains 20% F or 244.8 mg/mL of F F dosage = 1.0 mg/L as F Dilution factor = $244.8 \times 1,000 \div 1.0 = 244,800$ Maximum arsenic contribution = $60 / 244,800$ mg/L = 0.245 µg/L Normal arsenic contribution = $28 \div 244,800$ mg/L = 0.114 µg/L	0.114 µg/L (normal) 0.245 µg/L (maximum)
Bimetallic zinc metaphosphate	<2 mg/L	1.7 mg/L	Arsenic contribution = 2 mg/L x 1.7 mg/L	0.0034 µg/L
Potassium permanganate (KMnO ₄)	4.8 mg/L	0.35 mg/L	Arsenic contribution = 4.8 mg/L x 0.35 mg/L	0.00168 µg/L
Chlorine	All manufacturer reports indicate that arsenic is not present in gaseous chlorine.			0
Total arsenic contributed by treatment chemicals				0.279 µg/L (maximum)

*Based on dry equivalents.

Table 3. Arsenic contributed by chemicals used to treat groundwater at North Cheshire Wellfield

Treatment Chemical	Amount of Arsenic in Product	Dosage	Calculation of Contribution	Arsenic Contribution
Sodium hypochlorite (NaOCl)	0.8 mg/L (maximum)	1.2 mg/L	1 lb of chlorine reacts with 1.128 lb of caustic soda to produce 1.05 lb of NaOCl. An excess of caustic soda is used as a stabilizer. Based on the arsenic concentration in the 50% caustic soda, the maximum arsenic concentration in the NaOCl is estimated to be 0.8 mg/L. Arsenic contribution = 0.8 mg/L x 1.2 mg/L	0.00096 µg/L
Fluorosilicic acid (H ₂ SiF ₆)	60 mg/L (maximum)	1.0 mg/L as F	Dilution factor = $244.8 \times 1,000 \div 1.0 = 244,800$ Maximum arsenic contribution = $60 \div 244,800$ mg/L	0.245 µg/L
Bimetallic zinc metaphosphate	<2 mg/L	1.7 mg/L	Arsenic contribution = 2 mg/L x 1.7 mg/L	0.0034 µg/L
Total arsenic contributed by treatment chemicals				0.249 µg/L (maximum)

Table 4. Maximum finished water arsenic concentrations based on chemical dosages applied in the treatment facilities

Treatment Chemical	Range of Chemical Dosage (mg/L)	Range of Maximum Arsenic Contribution (µg/L in finished water)
Sodium hydroxide	8.0-12.5	0.0156-0.024
Sulfuric acid	20	0.0002
Alum	10-80	0.00385-0.0308
Potassium permanganate	0.30-0.35	0.0014-0.00168
Ferric chloride	7	0.037
Synthetic polymer A	2.0	0.001
Synthetic polymer B	4.0	0.004
Chlorine	1.2-2.8	0.000
Sodium hypochlorite	1.2	0.00096
Bimetallic zinc metaphosphate	1.5-1.7	0.0030-0.0034
Fluorosilicic acid	1.0	0.245

Table 5. Maximum finished water arsenic concentrations based on chemical dosages applied in the treatment facilities

Treatment Facility	Trace Arsenic Concentration (µg/L)	
	Calculated Maximum	Analytical Result
Lake Gaillard WTP*	0.279	<1
Lake Saltonstall WTP	0.299	<1
West River WTP	0.306	<1
North Cheshire Wellfield	0.249	<1
All other wellfields (N=4)	0.248	<1

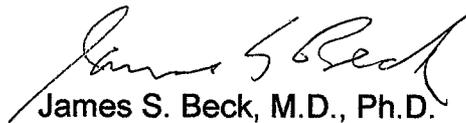
*Water treatment plant

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May 30, 2012

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7303 Young Road NW
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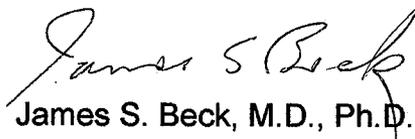
I am qualified as an expert in fluoridation by knowledge, skill, experience, training, or education and in my opinion, the bulk fluoridation products, fluorosilicic acid, sodium fluorosilicate, and sodium fluoride, are not "safe and effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water at 0.7 to 1.3 ppm of fluoride ion.



James S. Beck, M.D., Ph.D.
Professor Emeritus of Medical Biophysics
University of Calgary
Date: May 30, 2012

I am a physician and scientist (biophysics) and a co-author of a 2010 book on fluoridation. After studying fluoridation for a decade I was thoroughly convinced that it is not substantially effective in preventing cavities, that it constitutes a risk of harm particularly to special groups in any sizable population, and that it is a violation of the requirements of medical ethics.

Respectfully,



James S. Beck, M.D., Ph.D.

geraldsteel@yahoo.com

Gerald Steel

From: Kathleen Thiessen [kmt@senes.com]
Sent: Tuesday, June 05, 2012 2:04 PM
To: geraldsteel@yahoo.com
Subject: water fluoridation
Attachments: General statement (Thiessen).pdf; ATT00006.txt

Mr. Gerald Steel PE
Attorney at Law
7303 Young Road NW
Olympia WA 98502

Dear Mr. Steel:

I am a professional in the field of risk analysis, including exposure assessment, toxicity evaluation, and risk assessment. I have served on two subcommittees of the National Research Council's Committee on Toxicology that dealt with fluoride exposure and toxicity, including the NRC's Committee on Fluoride in Drinking Water. I have also authored an Environmental Protection Agency report on fluoride toxicity.

I am qualified as an expert in fluoride exposure, effects, and toxicity (including effects of water fluoridation) by knowledge, skill, experience, training, and education, and in my opinion, the bulk fluoridation products, fluorosilic acid, sodium fluorosilicate, and sodium fluoride, are not "safe and effective" to aid in the prevention and prophylactic treatment of dental caries disease when used to make fluoridated public drinking water at 0.7 to 1.3 ppm of fluoride ion.

Signed: Kathleen M. Thiessen, Ph.D.

Date: June 5, 2012

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General Comments
on the
Fluoridation of Drinking Water
for Prevention of Dental Caries

September 7, 2011

Kathleen M. Thiessen, Ph.D.
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The author of these comments is a professional in the field of risk analysis, including exposure assessment, toxicity evaluation, and risk assessment. She has recently served on two subcommittees of the National Research Council's Committee on Toxicology that dealt with fluoride exposure and toxicity, including the NRC's Committee on Fluoride in Drinking Water. She has also authored an Environmental Protection Agency report on fluoride toxicity.

These comments are not to be considered a comprehensive review of fluoride exposure or toxicity. Opinions and conclusions expressed herein are those of the author.

Summary. Although fluoridation of drinking water for the purpose of caries prevention is widely practiced in the United States and a few other countries, and is strongly encouraged by some governments and public health agencies, several important concerns have not been adequately addressed:

- (1) Available data do not support a role of community water fluoridation in improving dental health.
- (2) A variety of adverse health effects are associated with fluoride exposures.
- (3) By fluoridation of drinking water, governments and water suppliers are indiscriminately administering a drug to the population, without individual evaluation of need, appropriate dose, efficacy, or side effects.

These concerns are discussed in more detail below. Governments and health agencies that are serious about protecting the health of their populations should call for an immediate end to community water fluoridation.

(1) Available data do not support a role of community water fluoridation in improving dental health.

The U.S. Department of Health and Human Services (HHS) considers community water fluoridation to be important in the prevention of dental caries (Federal Register 2011), as do governments and health agencies in a few other countries. However, the question of whether water fluoridation actually produces a benefit requires further attention.

The University of York has carried out perhaps the most thorough review to date of human studies on effects of fluoridation. Their work (McDonagh et al. 2000) is often cited as showing the safety and efficacy of water fluoridation, but it actually does neither (Wilson and Sheldon 2006; Cheng et al. 2007). The report mentions a surprising lack of high quality studies demonstrating benefits, and also finds little evidence that water fluoridation reduces socioeconomic disparities:

Given the level of interest surrounding the issue of public water fluoridation, it is surprising to find that little high quality research has been undertaken. (McDonagh et al. 2000)

Water fluoridation aims to reduce social inequalities in dental health, but few relevant studies exist. The quality of research was even lower than that assessing

overall effects of fluoridation. (Cheng et al. 2007)

Evidence relating to reducing inequalities in dental health was both scanty and unreliable. (Wilson and Sheldon 2006)

The apparent benefit is modest, about a 15% difference in the proportion of caries-free children (McDonagh et al. 2000). The American Dental Association (2005) states that “water fluoridation continues to be effective in reducing dental decay by 20-40%,” which would translate to less than 1 decayed, missing, or filled permanent tooth (DMFT) in older children and adolescents (based on U.S. data from CDC 2005).

Neither McDonagh et al. (2000) nor the ADA (2005) mentions that fluoride exposure appears to delay the eruption of permanent teeth, although this has been known since the 1940s (Short 1944; NRC 2006). A delay in tooth eruption alters the curve of caries rates with respect to age and complicates the analysis of age-specific caries rates (Psoter et al. 2005; Alvarez 1995; Alvarez and Navia 1989). Specifically, “the longer the length of exposure to the oral environment the greater is the risk of the tooth becoming carious” (Finn and Caldwell 1963; citing Finn 1952). Komárek et al. (2005) have calculated that the delay in tooth eruption due to fluoride intake may explain the apparent reduction in caries rates observed when comparisons are made at a given age, as is usually done.

Most studies of benefits of fluoride intake or fluoridation have failed to account for a number of important variables, including individual fluoride intakes (as opposed to fluoride concentrations in the local water supplies), sugar intake, socioeconomic variables, and the general decline in caries rates over the last several decades, independent of water fluoridation status. When World Health Organization data on oral health of children in various countries are compared, similar declines in caries over time are seen in all developed countries, regardless of fluoridation status (Cheng et al. 2007; Neurath 2005). The only peer-reviewed paper to be published from California's major oral health survey in the 1990s reported no association between fluoridation status and risk of early childhood caries (Shiboski et al. 2003). Several studies show differences in caries rates with socioeconomic status or dietary factors but not with fluoridation status (e.g., Adair et al. 1999; Hamasha et al. 2006).

In general, the role of diet and nutrition in good dental health seems to be underappreciated. For example, Cote et al. (2004) have documented a much lower rate of caries experience in refugee children from Africa than in U.S. children or refugee children from Eastern Europe, a situation that the authors attribute more to the amount of sugar in the diet than the presence of fluoride in the water. Finn (1952) provides an extensive review of dental caries in “modern primitive peoples,” concluding that they “show less dental caries than do most civilized peoples. . . . Evidence indicates, however, that primitive peoples have an increased caries attack rate when brought into contact with modern civilization and a civilized diet.”

A number of sources (reviewed by NRC 2006), including the Centers for Disease Control and Prevention (CDC 2001), indicate that any beneficial effect of fluoride on teeth is topical (e.g., from toothpaste), not from ingestion. Featherstone (2000) describes mechanisms by which topical fluoride has an anti-caries effect and states that “[f]luoride incorporated during tooth development [i.e., from ingested fluoride] is insufficient to play a significant role in caries protection.” Also:

The fluoride incorporated developmentally—that is, systemically into the normal tooth mineral—is insufficient to have a measureable effect on acid solubility. (Featherstone 2000)

The prevalence of dental caries in a population is not inversely related to the concentration of fluoride in enamel, and a higher concentration of enamel fluoride is not necessarily more efficacious in preventing dental caries. (CDC 2001)

Fluoride concentrations in drinking water or saliva are too low to be contributing significantly to a topical anti-caries effect, especially since most drinking water is not “swished” around the teeth before being swallowed. CDC (2001) states that “The concentration of fluoride in ductal saliva, as it is secreted from salivary glands, is low—approximately 0.016 parts per million (ppm) in areas where drinking water is fluoridated and 0.006 ppm in nonfluoridated areas. This concentration of fluoride is not likely to affect cariogenic activity.”

The single study that has examined caries experience in relation to individual fluoride intakes at various ages during childhood (the Iowa study) has found no association between fluoride intake and caries experience; caries rates (% of children with or without caries) at ages 5 and 9 were similar for all levels of fluoride intake (Warren et al. 2009). The authors state that “the benefits of fluoride are mostly topical” and that their “findings suggest that achieving a caries-free status may have relatively little to do with fluoride *intake*” (emphasis in the original). Most of the children with caries had “relatively few decayed or filled surfaces” (Warren et al. 2009). The authors' main conclusion:

Given the overlap among caries/fluorosis groups in mean fluoride intake and extreme variability in individual fluoride intakes, firmly recommending an “optimal” fluoride intake is problematic. (Warren et al. 2009).

The national data set collected in the U.S. in 1986-1987 (more than 16,000 children, ages 7-17, with a history of a single continuous residence) shows essentially no difference in caries rates in the permanent teeth of children with different water fluoride levels (Table 1; Fig. 1; data obtained from Heller et al. 1997; similar data can be obtained from Iida and Kumar 2009). Analysis in terms of mean DMFS (decayed, missing, or filled tooth surfaces) for the group (Fig. 2), as opposed to caries prevalence, shows an apparent 18% decrease between the low-fluoride (< 0.3 mg/L) and fluoridated (0.7-1.2 mg/L) groups. In absolute terms, this is a decrease of about one-half (0.55) of one tooth surface per child. One possible explanation is delayed tooth eruption, which was not considered in the study. Note that the mean DMFS for the highest fluoride group is higher than for either of the two intermediate groups, also indicating that DMFS scores are not solely a function of water fluoride concentration. When the data are examined by the distribution of DMFS scores (Fig. 3), no real difference in caries experience with respect to water fluoride concentration is observed.

The available data, responsibly interpreted, indicate little or no beneficial effect of water fluoridation on oral health.

(2) A variety of adverse health effects are associated with fluoride exposures.

For most of the U.S. population, the single largest source of fluoride exposure is municipal tap water, including tap water used directly, beverages and foods prepared with municipal tap water

either at home or in restaurants, and commercial beverages and processed foods prepared with municipal tap water. For a water fluoride level of 1 mg/L (1 ppm), which is the level still used in most fluoridated U.S. cities, estimated average exposures to fluoride from all sources range from about 0.03 mg/kg/day (mg of fluoride per kg of body weight per day) for adults and nursing infants to 0.09 mg/kg/day for non-nursing infants (especially infants fed formula prepared with fluoridated tap water). Note that these are estimated *average* exposures. For individuals with high tap water consumption (discussed by NRC 2006), total fluoride exposures can exceed 0.1 mg/kg/day for some adults and may reach 0.2 mg/kg/day for some infants. In one of the few studies to evaluate individual intake of fluoride from all sources, Warren et al. (2009) report individual fluoride intakes (from all sources) in excess of 0.2 mg/kg/day for some infants.

The NRC (2006) identified several sizeable subgroups of the U.S. population that require special consideration due to above-average fluoride exposures, increased fluoride retention, or greater susceptibility to effects from fluoride exposures. Groups known to be at risk of high fluoride intake include those with high water intake (e.g., outdoor workers, athletes, and individuals with diabetes insipidus or other medical conditions) or exposure to other sources of fluoride intake (NRC 2006). In addition, people with impaired renal function are at higher risk of adverse effects per unit intake of fluoride, due to impaired excretion of fluoride and consequent higher fluoride concentrations in the body. Tap water consumption varies among individuals by more than a factor of 10, depending on age, activity level, and the presence of certain health conditions such as diabetes insipidus (NRC 2006; see also Warren et al. 2009 for an example of estimated fluoride intakes for individual children at different ages). A substantial number of infants have water consumption rates in excess of 0.1 L/kg/day (100 mL per kg body weight per day; NRC 2006; EPA 2004a).

The U.S. Department of Health and Human Services (HHS) recently proposed a new recommendation regarding fluoride concentrations in drinking water (Federal Register 2011), the primary change being from a recommended range of 0.7-1.2 mg/L fluoride in drinking water (0.7-1.2 ppm) based on ambient local temperatures, to a single value of 0.7 mg/L (0.7 ppm), regardless of temperature. At the proposed fluoride concentration of 0.7 mg/L in drinking water, infants consuming at least 0.1 L/kg/day of tap water will have fluoride intakes at and above 0.07 mg/kg/day, and some will exceed 0.15 mg/kg/day (NRC 2006).

The HHS recommendation addresses only dental fluorosis (discussed below), while ignoring a long list of other health concerns for the U.S. population. Dental fluorosis itself has been associated with increased risks of various adverse health effects, including thyroid disease, lowered IQ, and bone fracture (Alarcón-Herrera et al. 2001; Zhao et al. 1996; Li et al. 1995; Lin et al. 1991; Desai et al. 1993; Yang et al. 1994; Jooste et al. 1999; Susheela et al. 2005). To the best of my knowledge, no studies in the U.S. or Canada have looked for associations between dental fluorosis and risk of other adverse effects. However, the failure to look for adverse health effects does not demonstrate the absence of adverse health effects.

The NRC (2006) indicated that the Environmental Protection Agency's (EPA's) present drinking water standards for fluoride (maximum contaminant level goal [MCLG] and maximum contaminant level [MCL], both at 4 mg/L) are not protective of human health, based on preventing severe dental fluorosis, stage II skeletal fluorosis, and increased risk of bone fractures. Given the wide range of water intake within the American population and the presence of other sources of fluoride intake, one can reasonably expect that a "safe" level of fluoride in

drinking water would be at least a factor of 10 below the “unsafe” level of 4 mg/L. EPA’s MCLG is defined as a “non-enforceable health goal which is set at a level at which no known or anticipated adverse effect on the health of persons occurs and which allows an adequate margin of safety” (EPA 2009). Dental fluorosis, skeletal fluorosis, and increased risk of bone fracture are all reasonably well known and acknowledged adverse health effects from fluoride exposure. However, EPA is also required to consider the “anticipated” adverse effects (which may occur at lower levels of fluoride exposure than the “known” effects) and allow for an adequate margin of safety. The proposed HHS recommendation for water fluoridation at 0.7 mg/L is not adequate to protect against known or anticipated adverse effects and does not allow an adequate margin of safety to protect young children, people with high water consumption, people with kidney disease (resulting in reduced excretion of fluoride), and other potentially sensitive population subgroups.

In addition to the “known” adverse health effects of dental fluorosis, skeletal fluorosis, and increased risk of bone fracture, “anticipated” adverse health effects from fluoride exposure or community water fluoridation include (but are not limited to) carcinogenicity, genotoxicity, endocrine effects, increased blood lead levels, and hypersensitivity (reduced tolerance) to fluoride. These effects (described in more detail below) are not as well studied as the dental and skeletal effects, which should indicate that a greater margin of safety is necessary to ensure protection of the population—“in the face of uncertain evidence it is important to act in a manner that protects public health” (Tickner and Coffin 2006). In addition, it should be noted that some of these effects may occur at lower fluoride exposures than those typically associated with dental or skeletal effects, such that protection against the dental or skeletal effects does not necessarily ensure protection against other anticipated adverse health effects. Elimination of community water fluoridation is the best way to reduce fluoride exposures for most individuals to a level at which adverse health effects are unlikely.

A few comments regarding the interpretation of the available fluoride studies may be helpful. As Cheng et al. (2007) have described, a “negative” study may simply mean that the study was not sufficiently sensitive to demonstrate a moderate (as opposed to large) effect. This is often due to use of too small a sample size. In addition, study populations are often grouped by community, water source, or fluoride concentration in the water, rather than by individual intake. Due to the wide variation in drinking water intake, this approach results in study groups with overlapping intakes and makes it difficult to detect dose response relationships that do in fact exist.

The few studies that have looked at age-dependent exposure to fluoride have found increased risks of adverse effects (e.g., Bassin et al. 2006 for osteosarcoma; Danielson et al. 1992 for hip fracture risk); studies that have not looked at age-dependent exposure cannot be assumed to provide evidence of no effect. Similarly, studies that have used a measure of current exposure where a cumulative measure would be more appropriate, or vice versa, cannot be assumed to demonstrate lack of an effect.

Studies of fluoride toxicity in laboratory animals are sometimes dismissed as irrelevant because the exposures or fluoride concentrations used were higher than those expected for humans drinking fluoridated tap water. It is important to know that animals require much higher exposures (5-20 times higher, or more; see NRC 2006; 2009) than humans to achieve the same effects or similar fluoride concentrations in bone or serum. In other words, humans are considerably more sensitive to fluoride than are most animal species that have been studied.

A number of adverse health effects can be expected to occur in at least some individuals when estimated average intakes of fluoride are around 0.05 mg/kg/day or higher (NRC 2006; 2009). For persons with iodine deficiency, average intakes as low as 0.01-0.03 mg/kg/day could produce effects (NRC 2006). The next few sections briefly summarize some (not all) of the adverse health effects, known and anticipated, that should be considered in any reevaluation of the drinking water standards for fluoride. Most of these effects have been reviewed in detail by the NRC (2006), although the NRC did not specifically evaluate health risks over the whole range of fluoride intakes or attempt to identify a “safe” level of fluoride exposure.

Dental fluorosis

The main reason for the change in fluoridation levels proposed by HHS is the prevention of dental fluorosis, a condition ranging from mild spotting of the teeth to severe pitting and staining. Dental fluorosis is caused by excessive fluoride ingestion during the early years of childhood, before the permanent teeth erupt. The HHS recommendation is intended to limit the risk of dental fluorosis while maintaining caries protection (Federal Register 2011). The most recent data indicate a fluorosis prevalence in the U.S. (all levels of severity) of 40.7% in 1999-2004 vs. 22.6% in 1986-1987 for children ages 12-15 (Beltrán-Aguilar et al. 2010). The proposed change in water fluoridation level will put the U.S. in agreement with Canada, which in 2009 recommended a fluoride concentration of 0.7 mg/L for all parts of the country (Health Canada 2009).

Based on the 1986-1987 data set (as reported by Heller et al. 1997), which included water fluoride concentrations, fluoridating at 0.7 mg/L can be expected to bring the fluorosis prevalence in the U.S. down to about 27%. Elimination of fluoridation entirely, for the whole population, would be expected to bring the fluorosis prevalence down to that of the current low-fluoride population (to around 13% based on Heller et al. 1997; Fig. 4).

The only U.S. study to have looked at dental fluorosis and individual fluoride intake at various ages (the Iowa study) reported that for children with fluoride intakes above 0.06 mg/kg/day during the first 3 years of life, fluorosis rates were as high as 50% (Hong et al. 2006b). As mentioned above, at a fluoride concentration of 0.7 mg/L in drinking water, many infants will have fluoride intakes at and above 0.07 mg/kg/day, and some will exceed 0.15 mg/kg/day (NRC 2006). Thus a large fraction of infants and young children fed formula made with fluoridated tap water can be expected to develop dental fluorosis even at a water fluoride concentration of 0.7 mg/L.

The National Research Council considers severe dental fluorosis to be an adverse health effect and reports the general consensus in the literature that both severe and moderate dental fluorosis should be prevented (NRC 2006). Health Canada (2009) considers moderate dental fluorosis to be an adverse effect. The Iowa study indicates that high fluoride intake during the first 2 years of life is most important with respect to development of dental fluorosis of the permanent maxillary central incisors (the “top front teeth”)—the teeth that most affect a person's appearance—although fluoride intake up to at least 4 years old was also important (Hong et al. 2006a). The American Dental Association has issued a brief statement to the effect that parents should not prepare infant formula with fluoridated water if they are concerned about the possibility of their child developing dental fluorosis (ADA 2007). This is an admission that

dental fluorosis is undesirable, and that fluoridated tap water is not “safe” for all individuals. The CDC (2005) reports a higher likelihood of moderate and severe fluorosis for minority and low-income children. While for a variety of reasons it is appropriate for governments and health agencies to encourage breastfeeding of infants, in many family situations breastfeeding is not possible (e.g., in cases of adoption or of ill-health or death of the mother). It is therefore essential that tap water be safe for use in infant formula, without putting infants at increased risk of dental fluorosis.

Skeletal fluorosis

Bone fluoride concentrations in the ranges reported for stage II and III skeletal fluorosis will be reached by long-term fluoride exposures of 0.05 mg/kg/day or higher (estimated from NRC 2006). Bone fluoride concentrations, radiologic changes, and symptoms are not clearly correlated (Franke et al. 1975), and most U.S. studies do not categorize cases by stage. Recent case reports include fluorosis attributed to excessive ingestion of tea or toothpaste (Whyte et al. 2005; Hallanger Johnson et al. 2007; Kurland et al. 2007). Most of the literature addresses high fluoride exposures over a few years; there has been essentially no investigation of effects of low exposures over many years and no effort to identify fluorosis of any stage in the U.S. “Arthritis” (defined as painful inflammation and stiffness of the joints) is the leading cause of disability in the U.S., currently affects at least 46 million adults in the U.S. (including 50% of the population > 65 years old), and is expected to affect 67 million adults in the U.S. by 2030 (CDC 2006). The possibility that a sizeable fraction of “bone and joint pain” or “arthritis” in U.S. adults is attributable to fluoride exposure has not been addressed, although it is plausible, given what is known about fluoride intakes.

Increased risk of bone fractures

The NRC (2006) concluded that lifetime exposure to fluoride at an estimated average daily intake of 0.08 mg/kg/day (average adult fluoride intake with water at 4 mg/L) is likely to result in higher bone fracture rates, and the available information suggests an increased likelihood of bone fracture for daily fluoride intakes of 0.05 mg/kg/day (average adult fluoride intake at 2 mg/L). The Agency for Toxic Substances and Disease Registry (ATSDR) has identified a chronic-duration Minimal Risk Level (MRL) for oral exposure to fluoride of 0.05 mg/kg/day, based on an increased risk of bone fracture (ATSDR 2003). The NRC's findings (NRC 2006) indicate that the ATSDR's MRL is not protective enough. The available studies consider fluoride intake only in terms of the concentration in the local drinking water, and most use fluoridated water (1 mg/L, corresponding to an average daily intake of 0.03 mg/kg/day for adults) as a control. Thus there is probably considerable overlap in exposures between groups, making effects more difficult to distinguish, and the entire dose response range of interest has not been well studied. The findings in humans are consistent with animal studies that have found increased brittleness of bones with increased fluoride exposure (Clark and Mann 1938; Turner et al. 1997; 2001).

Danielson et al. (1992) reported an increased relative risk for hip fracture in a fluoridated area of 1.27 (95% CI 1.08-1.46) for women and 1.41 (95% CI 1.00-1.81) for men. These authors reported a difference between women exposed to fluoride prior to menopause and those exposed

afterwards. For women exposed prior to menopause, the fracture risk was considerably higher than for those not exposed to fluoride. Many studies of fracture risk have not looked at age-specific exposure, or have involved women exposed only after menopause, when fluoride uptake into bone is probably substantially lower.

The Iowa study reported effects on bone mineral concentration and bone mineral density with average childhood fluoride intakes of 0.02-0.05 mg/kg/day (Levy et al. 2009). Linear correlation between dental fluorosis and risk of bone fracture has been reported for children and adults (Alarcón-Herrera et al. 2001; Fig. 5). Bone fracture rates in children in the U.S. may be increasing (e.g., Khosla et al. 2003), but fluoride exposure has not been examined as a possible cause or contributor.

Carcinogenicity

Three U.S. courts have found water fluoridation to be injurious to human health, specifically that it may cause or contribute to the cause of cancer and genetic damage (described in detail by Graham and Morin 1999). The NRC's committee on fluoride toxicology unanimously concluded that "Fluoride appears to have the potential to initiate or promote cancers," even though the overall evidence is "mixed" (NRC 2006). Referring to the animal studies, the committee also said that "the nature of uncertainties in the existing data could also be viewed as supporting a greater precaution regarding the potential risk to humans." The committee discussed the limitations of epidemiologic studies, especially ecologic studies (those in which group, rather than individual, measures of exposure and outcome are used), in detecting small increases in risk—in other words, the studies are not sensitive enough to identify small or moderate increases in cancer risk; therefore a "negative" study does not necessarily mean that there is no risk (see also Cheng et al. 2007).

While the NRC did not assign fluoride to a specific category of carcinogenicity (i.e., known, probable, or possible), the committee did not consider either "insufficient information" or "clearly not carcinogenic" to be applicable. The committee report (NRC 2006) includes a discussion of how EPA establishes drinking water standards for known, probable, or possible carcinogens; such a discussion would not have been relevant had the committee not considered fluoride to be carcinogenic. The question becomes one of how strongly carcinogenic fluoride is, and under what circumstances.

The case-control study by Bassin et al. (2006) is the only published study thus far to have looked at age-dependent exposure to fluoride. This study reported a significantly elevated risk of osteosarcoma in boys as a function of estimated age-specific fluoride intake. Osteosarcoma is a bone cancer that commonly results in amputation of an affected limb and may result in death. At the very least, this study indicates that similar studies of pediatric osteosarcoma that have not looked at age-dependent intake cannot be considered to show "no effect." A recent review of osteosarcoma risk factors (Eyre et al. 2009) lists fluoride among "a number of risk factors that emerge with some consistency" and considers fluoride exposure to have a "plausible" role in etiology of osteosarcoma.

While a few other studies (e.g., Gelberg et al. 1995; Kim et al. 2011) have looked at individual fluoride exposure (as opposed to group or ecologic measures of exposure), these have looked at total fluoride exposure until time of diagnosis or treatment. Given that there is a "lag time" of a

few years between onset of a cancer and its diagnosis, use of cumulative fluoride exposure until time of diagnosis is potentially misleading, as fluoride exposure during the last several years (during the “lag time”) cannot have contributed to the initiation of a cancer but could have a significant effect on the estimate of cumulative fluoride exposure.

The 1990 National Toxicology Program (NTP) study on sodium fluoride officially concluded that “there was *equivocal evidence of carcinogenic activity* of sodium fluoride in male F344/N rats, based on the occurrence of a small number of osteosarcomas in dosed animals” (NTP 1990; italics in the original). According to the published report, a “small number of osteosarcomas occurred in mid- and high-dose male rats. These neoplasms occurred with a significant dose response trend, but at a rate within the upper range of incidences previously seen in control male rats in NTP studies” (NTP 1990). It is important to realize that the historic controls from previous studies had not had the special low-fluoride diet used for this study, and therefore more properly constitute a low- to mid-range exposed group rather than a control group. This and other concerns were described in a memo within the Environmental Protection Agency (Marcus 1990) and reported in the press (Hileman 1990). These concerns and the testimony before the U.S. Senate of the union representing EPA scientists (Hirzy 2000) should be taken seriously.

In humans, osteosarcomas tend to occur most commonly in young people (pediatric cases) or the very old (adult or geriatric cases), with a higher incidence in males than in females (Bassin et al. 2006). Sergi and Zwerschke (2008) indicate that 60-75% of cases are in patients between 15 and 25 years old. In the NTP 2-year study, fluoride exposure was begun when the animals were 6 weeks old, as is typical for NTP and similar studies (Hattis et al. 2004). Puberty in the rat typically occurs at about 32 days of age in females and 42 days in males (e.g., Gray et al., 2004; Evans 1986). Thus, the age of 6 weeks in the NTP study probably corresponds to pubertal or post-pubertal animals. The cases of osteosarcoma in the rats were reported in the late stages of the test, and probably corresponded to geriatric osteosarcomas in humans. In Bassin’s study, the age range for which the fluoride-osteosarcoma association was most apparent was for exposures at ages 4-12 years, with a peak for exposures at age 6-8 years (Bassin et al. 2006). Very likely, the fluoride exposures in most of the animal studies have started after the age corresponding to the apparent most susceptible age in humans, and thus these animal studies may have completely missed the most important exposure period with respect to initiation of the majority of human osteosarcomas. Therefore, this animal study cannot be interpreted as showing no evidence of causation for pediatric osteosarcoma, although, properly interpreted, it does show evidence for causation of geriatric osteosarcoma.

Genotoxicity

Genotoxicity, or the ability to damage the genetic material (genes and chromosomes) of cells, is considered indicative of potential carcinogenicity. A number of mammalian *in vitro* systems have shown dose-dependent cytogenetic or cell transformational effects from fluoride exposure (reviewed by NRC 2009). Several reports suggest an indirect or promotional mechanism, e.g., inhibition of DNA synthesis or repair enzymes, rather than a direct mutagenic effect (Lasne et al. 1988; Aardema et al. 1989; Aardema and Tsutsui 1995; Meng and Zhang 1997). Human cells seem to be much more susceptible to chromosome damage from fluoride than are rodent cells (Kishi and Ishida 1993).

A recent paper by Zhang et al. (2009) describes a new testing system for potential carcinogens, based on induction of a DNA-damage response gene in a human cell line. Sodium fluoride tests positive in this system, as do a number of other known carcinogens, representing a variety of genotoxic and nongenotoxic carcinogenic mechanisms. Known noncarcinogens—chemicals not associated with carcinogenicity—did not test positive. The system described by Zhang et al. (2009) is considerably more sensitive than the older systems for most chemicals examined; a positive effect was seen at a fluoride concentration of about 0.5 mg/L, or a factor of 10 lower than in other systems.

A fluoride concentration of 0.5 mg/L in urine will routinely be exceeded by many people consuming fluoridated water (NRC 2006); for people with substantial fluoride intake, serum fluoride concentrations may also reach or exceed 0.5 mg/L. Acute fluoride exposures (e.g., accidental poisoning, fluoride overfeeds in drinking water systems) have resulted in fluoride concentrations in urine well in excess of 5 mg/L in a number of cases (e.g., Penman et al. 1997; Bjørnhagen et al. 2003; Vohra et al. 2008). Urine fluoride concentrations can also exceed 5 mg/L if chronic fluoride intake is above about 5-6 mg/day (0.07-0.09 mg/kg/day for an adult; based on NRC 2006). Thus, kidney and bladder cells are probably exposed to fluoride concentrations in the ranges at which genotoxic effects have been reported *in vitro*, especially when the more sensitive system of Zhang et al. (2009) is considered. Based on the results of Zhang et al. (2009), most tissues of the body are potentially at risk if serum fluoride concentrations reach or exceed 0.5 mg/L. In addition, cells in the vicinity of resorption sites in fluoride-containing bone are potentially exposed to very high fluoride concentrations in extracellular fluid (NRC 2006) and thus are also at risk for genotoxic effects.

Endocrine effects

The NRC (2006) concluded that fluoride is an endocrine disruptor. Endocrine effects include altered thyroid function or increased goiter prevalence (at fluoride intakes of 0.05-0.1 mg/kg/day, or 0.01-0.03 mg/kg/day with iodine deficiency), impaired glucose tolerance (at fluoride intakes above 0.07 mg/kg/day), a decrease in age at menarche in girls in fluoridated towns, and disruptions in calcium metabolism (calcitonin and parathyroid function, at fluoride intakes of 0.06-0.15 mg/kg/day or higher). ATSDR's toxicological profile for fluoride (ATSDR 2003) refers to an animal study of thyroid function that would give a lower MRL (value not given) than the MRL derived for bone fracture risk (0.05 mg/kg/day).

Thyroid dysfunction and Type II diabetes presently pose substantial health concerns in the U.S. (NRC 2006). Of particular concern is an inverse correlation between subclinical maternal hypothyroidism and the IQ of the offspring. In addition, maternal subclinical hypothyroidism has been proposed as a cause of or contributor to development of autism in the child (Román 2007; Sullivan 2009). Steingraber (2007) has described the decrease in age at puberty of U.S. girls and the associated increased risk of breast cancer. Calcium deficiency induced or exacerbated by fluoride exposure may contribute to other health effects (NRC 2006).

Increased blood lead levels

An increased likelihood of elevated blood lead levels is associated with use of silicofluorides (usually H_2SiF_6 or Na_2SiF_6) as the fluoridating agent (NRC 2006; Coplan et al. 2007). Approximately 90% of people on fluoridated water are on systems using silicofluorides (NRC 2006). The chemistry and toxicology of these agents, especially at low pH (e.g., use of fluoridated water in beverages such as tea, soft drinks, or reconstituted fruit juices), have not been adequately studied (NRC 2006). Associations between silicofluoride use and biological effects in humans have been reported, in particular, elevated levels of blood lead in children and inhibition of acetylcholinesterase activity (reviewed by Coplan et al. 2007). A recent study in rats found significantly higher concentrations of lead in both blood and calcified tissues of animals exposed to both silicofluorides and lead (Sawan et al. 2010).

In addition to biological effects of silicofluorides, the interaction of silicofluorides (as the fluoridating agent) and disinfection agents (specifically, chloramines) also increases the leaching of lead from plumbing fixtures into drinking water (Maas et al. 2005; 2007). For example, the interaction of silicofluorides and chloramines is the probable explanation for the high lead levels in drinking water and children's blood in Washington, D.C. a few years ago (Maas et al. 2005; 2007; Leonnig 2010). EPA considers lead to be a probable human carcinogen and to have no practical threshold with respect to neurotoxicity (EPA 2004b)—in other words, there is considered to be no safe level of lead exposure, and the MCLG for lead is zero (EPA 2009).

Additional adverse health effects

Fluoride intake is likely to affect the male reproductive-hormone environment, beginning at intakes of around 0.05 mg/kg/day (reviewed by NRC 2009). A “safe” intake with respect to male reproductive effects is probably somewhere below 0.03 mg/kg/day.

Grandjean and Landrigan (2006) list fluoride as an “emerging neurotoxic substance” that needs further in-depth studies. The major concern is neurotoxic effects during human development. The NRC (2006) concluded that “it is apparent that fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means.” A number of studies indicate an association of fluoride exposure with lower IQ in children (reviewed by NRC 2006; Connett et al. 2010).

The NRC has reviewed the possible association between exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) and increased risk of Down syndrome (trisomy 21) in children of young mothers, discussed a possible mechanism, and recommended further study (NRC 2006). Fetuses with Down syndrome are less likely to survive to birth, due both to higher natural fetal loss and to a high rate of pregnancy termination (Buckley and Buckley 2008; Forrester and Merz 1999; Siffel et al. 2004; Biggio et al. 2004).

Hypersensitivity or reduced tolerance to fluoride has been reported for exposure to fluoridated water (approximately 0.02 mg/kg/day for adults) or use of fluoride tablets (approximately 1 mg/day). Symptoms include skin irritation, gastrointestinal pain and symptoms (nausea, vomiting, diarrhea, constipation), urticaria, pruritus, stomatitis, chronic fatigue, joint pains, polydipsia, headaches, and other complaints (Waldbott 1956; 1958; Feltman and Kosel 1961; Grimbergen 1974; Petraborg 1977; Spittle 2008; reviewed by NRC 2006). Patients were often

unaware that their drinking water contained fluoride. Symptoms improved with avoidance of fluoridated water and recurred with consumption of fluoridated water or with experimental challenge with sodium fluoride. Double-blind tests of patients have confirmed hypersensitivity to fluoride (Grimbergen 1974; Waldbott 1956; 1958). Many of the observed symptoms represent true allergic phenomena, while others (e.g., gastrointestinal symptoms) could be due to a lower level of tolerance for fluoride (intoxication at lower exposure; Waldbott 1956; 1958).

(3) By fluoridation of drinking water, governments and water suppliers are indiscriminately administering a drug to the population, without individual evaluation of need, appropriate dose, efficacy, or side effects.

The U.S. Food and Drug Administration (FDA) considers fluoride in toothpaste to be a non-prescription drug (e.g., FDA undated-a; undated-b) and fluoride “supplements” (usually tablets or lozenges) to be prescription drugs (e.g., Medline Plus 2008). Most prescription fluoride supplements are considered unapproved drugs (for example, see DailyMed 2011a,b,c), meaning that they “may not meet modern standards of safety, effectiveness, quality, and labeling” (FDA 2011). The goal of community water fluoridation is to provide a dental health benefit to individuals and to the population generally (Federal Register 2010), and EPA's recent reference (Federal Register 2010) to a “treated population” acknowledges this use of drinking water systems to deliver a drug to entire populations. This in effect puts local governments and water treatment personnel in charge of administering a chemical (i.e., a drug) to the population in an effort to improve individual and population health (Cross and Carton 2003; Cheng et al. 2007). Many people consume more fluoride from tap water than from either non-prescription (toothpaste) or prescription (tablets or lozenges) fluoride sources, without any monitoring for either efficacy or side effects, without the “drug information” or warning labels generally provided for drugs, and without any semblance of informed consent.

In addition, most fluoridation operations use fluorosilicates (usually H_2SiF_6 or Na_2SiF_6) rather than sodium fluoride (NaF). The chemistry and toxicology of these compounds have not been adequately studied, although important differences in biological effects between silicofluorides and simple fluorides (e.g., NaF) have been reported (Coplan et al. 2007; NRC 2006; Masters et al. 2000; Masters and Coplan 1999). The NRC (2006) discussed the increased toxicity of aluminofluorides and beryllifluorides vs. fluoride alone, as well as the different mechanisms of action of the different chemical combinations. It is irresponsible to recommend addition of fluoride, or a particular concentration of fluoride to be added, without a comprehensive review of the substances (H_2SiF_6 or Na_2SiF_6) that are actually added. In addition, fluoridation chemicals often contain impurities such as lead and arsenic, for which EPA has set MCLGs of zero (EPA 2006), such that a water supplier is actually adding contaminants for which the ideal maximum amount in drinking water is zero.

In summary, it is irresponsible to promote or encourage uncontrolled exposure of any population to a drug that, at best, is not appropriate for many individuals (e.g., those who do not want it, those whose water consumption is high, formula-fed infants, people with impaired renal function) and for which the risks are inadequately characterized and inadequately disclosed to the public. Elimination of community water fluoridation at the earliest possible date would be in the best interest of public health.